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EMOTION AND PERSONALITY

VOLUME II Neurological and Physiological Aspects

Emotion and Personality

VOLUME II NEUROLOGICAL AND PHYSIOLOGICAL ASPECTS

By Magda B. Arnold

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PREFACE

This work, which has been divided into two volumes for the sake of greater convenience to the reader, represents an attempt to develop a theory of emotion and feeling that can adequately integrate the psychological, neurological, and physiological aspects of affective phenomena and place them in their proper perspective as important factors in personality integration.

Thus far, theories of emotion that have taken account of neurological or physiological findings have started from some particular piece of evidence from these fields and have attempted to work out the psychological implications (for instance, Cannon's emergency theory and thalamic theory, Papez's theory based on the hippocampal circuit, Hebb's theory based on reverberating circuits in the brain). In contrast, it will be demonstrated here that a phenomenological analysis of emotional experience can guide us in identifying the brain structures and pathways that mediate feelings and emotions. The resulting theory of brain function is not a speculative "model" that has only the most tenuous connections with experimental evidence. Rather, the brain circuits mediating various psychological activities have been worked out link by link from a vast array of research studies culled from many fields until a well-articulated structure could be discerned. It is true that some neural connections are not fully documented and some details are missing that may turn out to be crucial. But there is a hope that such an effort at integrating a vast and chaotic field, though it may be deemed premature, may prove its value by giving direction to at least some research that otherwise might have been haphazard. Psychologists no less than other scientists feel the urge to know the how and why of things; they may distrust what seems a deceptively simple answer but they cannot be content to have the answer delayed indefinitely. The theory here sketched may not be in accord with currently fashionable ways of looking at things, but it does integrate psychological, neurological, and physiological data in a consistent way.

In the first volume a historical review of theories of feeling and emotion was provided, together with a theory that connects emotion with motivation, interests, and attitudes. The present volume attempts to review available neurological and physiological research findings to discover whether they support the theory outlined in the first volume.

In Part I of this volume, various neurological theories of emotion are reviewed and evaluated (Ch. 1); in Chapters 2 to 4 the circuit that seems to mediate the sequence of psychological activities from perception to emotion and action is sketched in detail. Part II continues the description of the neurophysiological state in emotion: Chapter 5 deals with the electrical activity of the brain and its psychological significance, and Chapter 6 spells out the pattern of psychological, neural, and hormonal reactions that is characteristic of various instinctive and emotional actions. Chapter 7 evaluates the evidence for the physiological changes that take place during emotion, while Chapter 8 views the significance of these changes in the light of the literature on stress and homeostatic regulations. Thus Parts I and II show that the theory of emotion and feeling developed in the first volume is abundantly supported by neurological and physiological evidence. Since this evidence is presented in detail, it is possible to check every link of the proposed brain circuit connecting sensory and motor areas, cortex and subcortex. Part III, finally, shows how emotion fits into a theory of personality (Ch. 9), demonstrates that it is a factor in the formation of the self-ideal (Ch. 10), and discusses the role of the self-ideal in human conduct. The final chapter takes up some positive human emotions and their role in personality organization. The problem of measurement is discussed in the Appendix, which also reviews some experimental studies that bear on the connection of emotion and personality.

This work has been made possible by two fellowships that have given me the necessary leisure to begin it and, some years later, to complete it. My grateful thanks are due to Radcliffe College for awarding me the Helen Putnam Fellowship for Advanced Research, which gave me two happy and productive years at Harvard; and to the Guggenheim Foundation for a Guggenheim Fellowship, which made it possible to take a year's leave of absence when it was most useful.

During the arduous years of preparing the manuscript, there have been many who have given unstintingly of their time and interest. While I was in Cambridge, Professor Gordon W. Allport, Dr. J. G. Beebe-Center, Dr. Stanley Cobb, and Dr. J. C. Laidlaw, all of Harvard University, were my main advisers. Later, in Chicago, Dr. Percival Bailey and Dr. Frederick A. Gibbs, of Illinois University Medical School, were good enough to read the relevant chapters in the manuscript; Dr. Wendell J. S. Krieg and Dr. Ray S. Snider, of Northwestern University Medical School, gave me the opportunity to discuss with them some problems in their own fields. Dr. W. R. Hess, of Zurich, Switzerland, read several chapters and expansively discussed some of the points that were raised during two memorable visits, one to his country house and another to his town house. Dr. Paul D. MacLean, of the National Institute of Mental Health, at Bethesda, Maryland, read an earlier version of the first several chapters in the second volume and kindly drew my attention to recent work in his field with which I had not been previously acquainted. To all of them I am more grateful than I can say. Most of all, I owe a debt of gratitude to a colleague and helper in a former work, the Reverend J. A. Gasson, S.J., of Spring Hill College, Mobile, Alabama. He graciously and patiently read each chapter as it was written, criticized it, and encouraged me, often drew my attention to problems I had glossed over, and was always willing to discuss a difficult point until a feasible solution was reached. Without his steadfast encouragement I might have given up trying long before such consistency as I have achieved was possible.

I want also to thank the Widener Library and the Library of the Medical School at Harvard University for granting me staff privileges during my stay in Cambridge, as well as the Dean of the Northwestern University Medical School for his generous permission to use the library facilities there. Finally, I am grateful to the head of my department, the Reverend V. V. Herr, S.J., at Loyola University in Chicago, for his unfailing consideration during the years spent in preparing this work.

MAGDA B. ARNOLD

Chicago
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Part I. BRAIN MECHANISMS IN EMOTION

1. NEUROLOGICAL THEORIES OF EMOTION

We saw in Chapters 9 and 10 of the preceding volume on the psychological aspects of emotion and personality that emotion is a complex process which starts when something is perceived and appraised. The appraisal arouses a tendency toward or away from the thing that is felt as emotion and urges to action. When we consider the whole process as it operates in the living being, it is clear that it must be mediated by neural excitation which produces both the psychological and physiological changes observed during emotion.

The James-Lange theory had proposed that emotion is simply the perception of physiological changes. With such a theory no special neural mechanism is required because sensory pathways are sufficient to explain emotional experience. Of course, the physiological changes perceived as emotion must first be produced, but James assumed that this occurs because the emotional situation has a "physical effect upon the nerves" (see Vol. I, Ch. 6). We have shown that the James-Lange theory is inadequate from a psychological point of view. It is also not adequate to explain the neurological evidence that has accumulated since the days of James and Lange.

W. B. Cannon (1927, 1931) was the first to make a careful survey of the experimental evidence in this field, including his own results, and to present a theory that was to correct the inadequacies of the earlier view.

CANNON'S CRITICISM OF THE JAMES-LANGE THEORY

First of all, Cannon listed the evidence showing that emotional experience and expression cannot be explained as the perception of sensations; he found five salient objections.

1. Behavior after the exclusion of the viscera. Sherrington had transected the spinal cord of dogs and their vagus nerve so that visceral nerves could no longer be excited from the brain. Yet these dogs barked, growled, and snapped when provoked to anger exactly as they had before the operation. Cannon and his associates removed the sympathetic nervous system from cats. During their long survival, the cats showed the same signs of anger or fear as they had before the operation. Later critics, notably Newman *et al.* (1930), objected that these animals did not necessarily *feel* emotion; they merely showed a change in behavior pattern that would have resulted in emotion had the peripheral nervous system been intact.

Cannon as well as his critics agreed that perception sets off some pattern of excitation that starts out from the brain and is relayed to the periphery. But Cannon claimed that the only part of the bodily mechanism necessary for experiencing emotion is the brain, which sets off the pattern, while James, Lange, and their followers insisted that this pattern must be registered in the periphery and result in visceral and circulatory changes before emotion can be experienced. Newman and his associates, on the other hand, emphasized the entire sequence of excitation from brain to periphery as equally necessary:

> Cortical, thalamic, organic and peripheral conditions are all necessary. Of utmost importance is the interpretation of the situation, an insightful response which determines whether or not the emotional pattern will be complete and therefore whether peripheral conditions shall function in emotion or in some non-emotional response. In short, the emotion is a configurational response concerned with the process of resolving a tension toward a particular goal. The qualitative aspects of this response inhere in its organization. (1930, p. 326)

This implies that the determining factor, the final criterion of emotion, is the interpretation of the situation and not the feel of the physiological changes—a far cry from James's position. If the emotion is "a configurational response concerned with . . . resolving a tension toward a particular goal," it is difficult to deny that Sherrington's dogs and Cannon's cats experienced emotion, because they interpreted the situation in the same way normal animals do, they showed the same goal and the same qualitative organization of their response. No doubt some rather intense sensations were missing because the visceral and circulatory changes could no longer occur, but the determining factor (the "insightful response") was present.

2. The visceral changes. Cannon's next point was that ***the same visceral*** *changes occur in all emotions.* Accordingly, these changes cannot provide the emotional experience which is qualitatively different in different emotions. Cannon here referred to his own investigations, according to which the same visceral changes occur in fear and in rage, during hypoglycemia and chills and fever, all induced by adrenaline secretion and sympathetic excitation.

Actually, if the physiological state were exactly alike in all emotions, that would make difficulty not only for the James-Lange theory but for Cannon's theory as well. The physiological state is perceived, if not as emotion then as organic sensation. If it were always the same, our organic sensations could not inform us that we are chilly or feverish rather than emotional. Yet we rarely mistake a chill for an emotion. In recent years it has been shown that different emotions are actually accompanied by different patterns of physiological changes, a finding that was anticipated by psychological analysis (see Arnold, 1945). We shall discuss the available evidence in a later chapter when we discuss Cannon's emergency theory of emotion (see Ch. 7).

3. Insensitivity of the viscera. Cannon pointed out that the viscera proper (stomach, intestines, liver, pancreas, spleen, kidneys, etc.) have fewer sensory nerves than other structures. Consequently, we are unaware of normal physiological processes occurring there, while some visceral sensations we do perceive are really sensations from surrounding structures.

The viscera, however, are not as insensitive as Cannon claimed. Dysmenorrhea and abdominal colic are the result of muscle cramps in the viscera and do not depend to any appreciable extent on surrounding structures. Crushing of the viscera is one of the means of producing experimental surgical shock, even under anesthesia; hence there must be sensory nerves relaying excitation to the brain. Abdominal operations under local anesthesia (e.g., excision of part of the stomach or intestines) are also extremely unpleasant, even though there may not be actual pain.

4. Rate of visceral changes. Cannon pointed out that smooth muscles have a comparatively long latency period (from 0.25 second in the cat to 0.85 second in the dog and 0.80 second in the horse). To this must be added the time of conduction from cortex to viscera and back. In addition, it takes some time to build up a peak of muscular contraction sufficient to be distinguished from the resting state. The reaction time for emotional responses to pictures of men and women was found by Wells to be about 0.80 second, and therefore is much shorter than it could be if emotional experience had begun only after the sensations from the viscera were perceived. Newman *et al.* retorted that the Wells experiment demonstrated simple sensory feeling and not emotion. In their own experiments these authors found that emotions were slow in developing:

> Our observers were able to report a type of experience that came almost immediately with the meaning of the presentation which was probably that which Wells observed, and yet there was an additional feature, a developing experience that came *only from three to fifteen seconds after the exposure of the material.* One observer reported that the experience was more vivid from two to three minutes later than it was at the time of the actual presentation. (1930, p. 313; Newman *et al.*'s italics)

From such reports the authors concluded that there was enough time for the slowly developing visceral reaction to be experienced as gradually increasing emotion. But what is this "type of experience that came almost immediately"? It surely is not merely cognitive. An example from Lehmann will make this clear: Supposing a thousand people read the announcement of Mr. X's death in their morning paper. They all have a cognitive experience; but the few who knew him and were fond of him will also have an emotional experience. It will come as a sudden pang, an emotional shock, for Mr. X's friends but not for anybody else. There is evidence for this kind of shock experience among the reports quoted by Newman and associates: "I saw the object and recognized that it was a face. Then it seemed to change and I realized what was wrong with it. At this time I got a distinct shock which I reported" (1930, p. 314). (This subject's reaction time was about .9 second.) Now there seems hardly any doubt that the emotion began when the subject realized that something was wrong with the face, nor that the shock experience was a genuine emotional reaction. We may refuse to call it a *complete* emotional experience and may agree with Newman that normally a whole sequence of bodily changes will follow which are all gradually sensed, so that the peak of the experience may not come for some time. But it remains a fact that for most of the people who read Mr. X's death notice that process never started. Hence the one absolutely necessary condition for an emotion is a realization of *what this means to me* as an individual: the death of a friend means that I will never be able to see him again, to talk to him, to be with him. In common experience, the emotion comes as soon as I realize how a given situation affects me: whether it means harm or loss or good fortune—even though the full impact of that realization may develop only gradually, very likely as the consequence of the various bodily changes that come with the emotion. For this reason, Newman's observer could report that the experience was more vivid two or three minutes after seeing the picture—but he did not say it was not present immediately.

In addition, there are many experimental investigations showing that autonomic changes come after the reported emotion. These are summarized at length in Lehmann's exhaustive treatise. There seems to be no reasonable doubt that the bodily changes in emotion occur later than the emotion itself.

5. *Effect of artificial induction of visceral changes.* Cannon here referred to the effect of adrenaline injections which produce a large number of visceral changes similar to those experienced in fear, yet arouse no emotion, according to subjective reports. Newman and his associates admitted the fact but insisted that such injections reproduce only part of the emotional pattern, and not the complete emotion. "There is no goal, nothing to be afraid of, joyful about, or angry at," they said; what wonder that there is no emotion? But it is only fair to point out, as Cannon did, that for James it was the *objectless* emotion which was the critical proof "that the immediate cause of emotion is a physical effect on the nerves" (quoted by Cannon, 1931, p. 288).

If Newman *et al.* saw the critical feature in the object-subject relation, they were departing widely indeed from the theory they sponsored. If the cumulative effect of all the peripheral changes *is* the emotion, as James thought, adrenaline should evoke a true emotional experience. Again, as we saw before, the only real difference between the two views is the factor considered *necessary* for an emotion. When, on the other hand, Cannon mentions a case reported by Dana—of a forty-year-old woman who had broken her neck at the third and fourth cervical level, was completely paralyzed and anesthetic, with the skeletal system practically eliminated and the sympathetic system completely so, yet showed emotions of grief, joy, displeasure, and affection exactly as before the accident—his evidence must be admitted as contradicting any peripheral theory of emotion. True, the patient's emotions could not have had the sensory reverberation they normally had, yet what really mattered, the realization of loss, or of the presence of someone loved, or of an interference with her plans, was present and sufficient to make her act and feel grief-stricken, joyful, loving, or displeased.

In summary, Cannon's criticism of the James-Lange theory has shown that the available evidence contradicts the theory on the following points: (1) Excluding the viscera from cortical connections does not alter emotional experience. (2) Visceral changes are too slow to be a source of emotion. (3) Artificial induction of visceral changes that are typical of emotion does not produce it. The two additional points made by Cannon, that the same visceral changes occur in very different emotional states and that the viscera are relatively insensitive structures are either doubtful for psychological reasons or contradicted by factual evidence.

CANNON'S THALAMIC THEORY

Cannon's own explanation of the neural mechanism in emotion is known as the *thalamic theory*. According to this theory, incoming sensory impulses receive an added "emotional quale" in their transit through the thalamus. This quality of emotion is, however, neither experienced nor expressed so long as there is cortical inhibition, an inhibition which is assumed to be present during the normal unemotional state. In an emotional situation, disinhibitory impulses from the cortex release the appropriate thalamic pattern which not only discharges over the peripheral nerves (somatic expression) but also excites nervous impulses to the cortex which give rise to emotional experience. According to Cannon:

> An external situation stimulates receptors and the consequent excitation starts impulses towards the cortex. Arrival of the impulses in the cortex is associated with conditioned processes which determine the direction of the response. Either because the response is initiated in a certain mode or figure and the cortical neurones therefore stimulate the thalamic processes, or because on their centripetal course the impulses from the receptors excite thalamic processes, they are roused and ready for discharge. That the thalamic neurones act in a special combination in a given emotional expression is proved by the reaction patterns typical of the several affective states. These neurones do not require detailed innervation from above in order to be driven into action. Being *released* for action is a primary condition for their service to the body—they then discharge precipitately and intensely. . . . When these neurones discharge in a particular combination, they not only innervate muscles and viscera but also excite afferent paths to the cortex. . . . The peculiar quality of the emotion is added to simple sensations when the thalamic processes are roused. (1927, p. 120)

This seems to mean that the thalamic processes are either *roused* directly by sensory impulses on arrival in the thalamus, as in the case of startle, or are *released* by cortically conditioned responses. A man who is afraid of riding in cars because he has had an accident, for instance, could feel fear at the sight of a car because it would arouse the memory of what had happened before (a "conditioned process"?) which had been followed by fear. In the case of startle, evidently the thalamic excitation is so strong that it overcomes cortical inhibition.

Now what was the evidence that convinced Cannon that emotion must be a release phenomenon? He mentioned the following points:

1. Decorticated animals exhibit exaggerated rage reactions (sham rage). If the thalamus is removed, the reaction disappears.
2. Unilateral lesions in the thalamic region add a marked affective element to sensations from the damaged side of the body.
3. A tumor affecting one side of the thalamus in man results in unilateral emotional expression, although cortical control of facial muscles is preserved bilaterally.
4. "Temporary impairment of cortical control of lower centers in light anesthesia, or permanent impairment by disease, releases free and often prolonged weeping and laughing." (1931, pp. 283–84)

Now let us see whether the evidence really demands a release theory of emotion. In the first place, Bard showed in 1928 that the thalamus is not necessary for the expression of sham rage. Typical rage occurred even after the thalamus and most of the hypothalamus were removed, provided only that the posterior hypothalamus and the brain stem were intact (Bard, 1950). It can be assumed, therefore, that the posterior hypothalamus and neighboring midbrain structures are necessary for the *expression* of rage. But such experiments do not prove that the hypothalamus generates rage, as has sometimes been assumed. Cannon himself always spoke of the thalamus as responsible for emotional *experience* by virtue of a relay to the cortex and for emotional *expression* by virtue of relays to the periphery.

Sham rage as disinhibition. The concept of cortical inhibition of lower structures seems to date back to Hughlings Jackson's dictum that a lesion cannot produce a gain in function. If such a gain occurs, it must be the result of disinhibition: the lesion must have destroyed a suppressor mechanism. Now the so-called "sham rage" was considered such a "gain in function" after cortical destruction; and Cannon assumed that it appeared because a suppressor mechanism was destroyed. "Sham rage" was observed in decorticated cats tied to the animal board as they came out of anesthesia. According to Cannon, sham rage is more intense and more frequent than rage in the normal animal, and apparently is a "gain in function."

But is sham rage more intense, more frequent, or more lasting than rage shown by the normal animal? According to Bard (1934) the normal animal also shows intense rage when it comes out of anesthesia and finds itself tied to the animal board. Neither is sham rage more lasting. It stops immediately the stimulus is withdrawn, while "it is sometimes not safe to approach normal animals for hours afterwards" (Bard, 1934). Sham rage is not more frequent than the real article because it is restricted to the occasions when the animal is subjected to pain or restraint.

It is true that for some time after the operation the animal seems to show rage at the slightest provocation, for instance, at a slight pinching of the tail. But Bard himself pointed out that such response may be the result of an irritation of nervous tissue. To decide between disinhibition and irritation as a cause of rage in decorticated cats, Bard and Rioch (1937) studied the behavior of such cats during a lengthy survival time and concluded that sham rage is not an irritation phenomenon.

However, the report does not give clear proof that the sham rage remains excessive. Out of four cats, one animal showed considerable fluctuation in the rage response (No. 103). Two more showed rage only on the most intense stimulation, when strapped to the animal board or pinched severely (No. 228, No. 244). Only one cat (No. 313) became enraged easily and intensely throughout its survival time, and this cat had been excessively wild before decortication. Kennard (1945) also reported that the sham rage shown by decorticated cats "became with time less frequent, less extreme and more difficult to elicit."

If rage were really a release phenomenon, decortication should induce a permanent emotional reaction that continues until the animal is completely exhausted. Instead, rage must be provoked after decortication and often requires more intense stimuli. Rage cannot be the result of thalamic release because it has been evoked in intact animals by electrical stimulation of the hypothalamus (Masserman, 1950; Hess, 1954), of the limbic and hippocampal cortex (Kaada *et al.*, 1953; Rothfield and Harman, 1954; and others), and even of the midbrain (Kelly *et al.*, 1946) and cerebellum (Zanchetti and Zoccolini, 1954).

Finally, removal of the cortex does not result in exaggerated fear, disgust, joy, or any other emotion. In fact, these emotions cannot be evoked in the decorticated animal. Besides rage, there is only one other emotion reported in decorticated cats. It consists in a sudden, precipitate running fit after intense sound stimulation. The cat starts running and dashes into walls and furniture until it is exhausted. These attacks seem to be quite similar to the sound-produced seizures in rats which have been described exhaustively during the last twenty years. Bard (1934a) called these running fits "fear" or "fright reactions," but if they are identical with the seizures observed in the rat, they seem to be related to startle rather than to fear, because they can be induced by sudden intense sound and can be prevented by fear or adrenaline injection (Arnold, 1944). These fright responses, like "sham rage," are massive explosive reactions. Completely undirected, they have only accidental similarity to genuine fear and genuine rage.

There is no evidence that emotion is generated either in the thalamus or the hypothalamus, or that it can be experienced and expressed when these structures are "released" from cortical inhibition. The evidence allows us to conclude that animals may react to restraint, pain, and intense sound even in the absence of the cortex. Such reactions seem to imply a short circuit between two relay stations, connecting sensory afferents with motor relays. Normally, these relays seem to be included in a longer circuit via the neocortex. The evidence does not justify the assumption that this longer circuit at one point switches from excitation to inhibition. The caudal hypothalamus and brain stem seem to be necessary for integrated expressions of fear and rage, but the thalamus and cortex seem to be necessary for the normal experience of these emotions.

Increased affectivity with thalamic lesions. Cannon's notion that unilateral thalamic tumors result in increased affectivity has been criticized by Lashley (1938). This critic carefully reviewed the whole set of observations, first reported by Head and later quoted by Cannon in support of the thalamic theory, and concluded that the so-called "changes in affectivity" are really changes in the intensity or quality of sensations: the pain is more intense, more burning, or more diffuse on the affected side. Lashley also pointed out that these changes in sensation are not confined to thalamic lesions. Increased sensitivity may occur when there is irritation of nerve tissue anywhere along the somatosensory pathway (e.g., in neuritis or syringomyelia). He concluded that such irritation produces sensations of pathological intensity or quality but not increased affectivity.

While it is true that any irritation of the somatosensory pathways will produce pain, such pain is a feeling and not a sensation, as we have tried to show in Chapter 4 of the preceding volume. At the same time, pain seems to be mediated not only by the thalamus (centrum medianum nucleus) but also by the posterior cingulate gyrus in the postcentral cortex; destruction of the centrum medianum nucleus alone will not prevent it. So far from inhibiting the thalamus which mediates this feeling, the cortex seems to add sharpness and intensity to the pain and makes it possible to locate the painful spot (see our discussion in the next chapter).

Unilateral emotional expression with unilateral thalamic lesions. When all emotional expression is abolished on the damaged side though the voluntary control of facial muscles is preserved, Cannon would postulate that the thalamic center relaying the expressive pattern to the periphery has been destroyed. But Bard has shown that this expressive pattern is retained as long as the hypothalamus is intact. This seems to imply that there are either two different emotional "centers," one in the thalamus, the other in the hypothalamus, or that emotional expression (but not necessarily emotional experience) is integrated at different levels of the nervous system. The thalamic theory allows for neither alternative.

Evidence from anesthesia and brain disease. When Cannon spoke of "temporary impairment of cortical control" in light anesthesia, or "permanent impairment of cortical control" in disease, he *assumed* that the cortex controls thalamic emotional expression, instead of *proving* it. Does light anesthesia really impair cortical control, i.e., does it impair cortical inhibition of emotion? True, the patient has no control over his movements, and his weeping or laughter does not seem caused by anything around him. But is such an emotion necessarily a "thalamic release"? The fact that a man is unconscious is no guarantee that there is no psychological activity. In sleep or during anesthesia, he can dream, and in his dream be afraid, enraged, or amused—and dreaming surely implies cortical functioning. Under nitrous oxide anesthesia, many patients actually have (and later remember) extremely vivid dreams (see W. James, 1920), and their weeping or laughter is merely the normal expression of their intense dream emotion. It is far safer to assume that weeping and laughing under anesthesia, as during sleep, are aroused by dream emotions even though the dream may be forgotten, than to assume that such emotional expression is the result of a "thalamic release," a "functional decortication." We know that people do forget dreams, yet everyone dreams (cf. Dement and

Kleitman, 1957). As long as even some people remember dreams under anesthesia and remember their dream emotions, we cannot maintain that the expression of emotion in that state is meaningless, the mechanical result of a temporary decortication.

The "permanent impairment of cortical control" by disease also cannot be proved. It is more likely that the prolonged laughter or weeping of patients with thalamic disease is aroused by the irritative stage of a degenerative process than that it is released by the destruction of an inhibitory mechanism. If the latter were the case, we should expect no emotion at all during the irritative stage because at that time the inhibitory mechanism would be stimulated. Emotional outbursts should appear only after tissue degeneration is complete, and then should be continuous and permanent because the inhibitory mechanisms would now be destroyed. Actually, the emotional outbursts appear early in the disease process and subside when it is halted.

This is shown, for instance, by Davison and Kelman's report (1939) that uncontrollable weeping and laughing occur with cortical as well as with thalamic lesions. Of the six cases of patients with cortical lesions who showed this symptom, at least four had no thalamic damage. When the tumor causing irritation was successfully removed, this symptom disappeared within five weeks after operation. In all these cases the patients laughed normally when amused and cried normally when perturbed. When a weeping or laughing spell began, they felt no emotion though some of them might begin to feel sad or happy during the course of the seizure. Such evidence is inconsistent with the notion that an inhibitory mechanism was destroyed by the disease. It is consistent with the idea that cortical and hypothalamic pathways can be excited either by an irritative process or by the appraisal of a situation as amusing or perturbing. In both cases, there will be emotional *expression,* but emotional experience will occur only when there is something to amuse or disturb the patient. When uncontrollable weeping brings on sadness, or uncontrollable laughter amusement, we may assume a secondary appraisal: If I weep, there must be something to weep about—and rationalization quickly finds it. If I laugh without cause, that in itself is funny.

Cannon's thalamic theory has appealed to many because it seems to support the notion that emotional drives are active continuously, if not in the conscious then in the unconscious; and the cortex was easily identified with the conscious, the thalamus with the unconscious. But there is no continuous emotional expression after decortication such as we would expect if decortication means the release of a thalamus that is generating emotions. In fact, we have seen that emotional expression in decorticated cats ends abruptly, as soon as the stimulus is withdrawn. Lashley (1938) pointed out that the brief emotional episodes in decorticated animals cannot compare with the persistent emotions found normally. Hence there is no evidence that the thalamus generates emotion and the cortex inhibits it.

The evidence seems to confirm Cannon's claim (as amended by Bard) of an area in the caudal hypothalamus that is necessary for the expression of fear and rage (cf. Ch. 6, Emotional Action Patterns), which is excited either directly by spinal afferents (in sham fear or sham rage) or via pathways from the cortex. But there are serious contradictions if it is assumed that these impulses are disinhibitory from cortex to thalamus and excitatory from thalamus to periphery. The contradictions disappear if we assume that the effector patterns are *excited* by cortical impulses.

There is actually direct evidence for cortical excitation of the thalamus during emotional expression. Dusser de Barenne and McCulloch (1938) pointed out that stimulation of the cortex always produces excitation of thalamic neurons, never inhibition. Moreover, recent experiments have shown that stimulation of the cortex can induce directed attack or flight (Kaada *et al.*, 1953; MacLean, 1949), which also speaks for the notion that emotion implies cortical excitation. We shall discuss these findings in detail in Chapters 3 and 6.

LATER NEUROPHYSIOLOGICAL EXPLANATIONS

Cannon's thalamic theory was the first attempt to suggest a brain mechanism which would account for emotional experience and expression. His theory did not specify any brain structure in particular but merely suggested that the cortex at large inhibits emotions while the thalamus produces them. Though there were flaws in the theory, it was a signal achievement at the time. Since then, neurological and neurophysiological research has gone on apace, discovering unsuspected functional relations. Though the evidence is anything but complete, theorists are bound to attempt some informed guess as to the nature of the brain mechanism that is activated during emotion. Advance in knowledge can come only with attempted explanations. If nothing is dared, nothing is won.

Obviously, every psychological experience is mediated by some neural structure. We know that sense experience is mediated by sensory cortical areas which receive impulses from the different receptor organs, though we do not know how the impulses arriving in these areas are translated into sense experience. We know that every movement is initiated by impulses from the motor area in the cortex, though we do not know how our decision to move is translated into neural impulses. Analagously, there should be some brain structure to mediate emotional experience and some brain structure to mediate emotional expression.

Since the way in which a mechanism functions is bound to give us some clue to understanding it, let us restate what our analysis of emotion discovered in Chapter 9 of the preceding volume:

> Something must be known (actually or in imagination) before it can be wanted, feared, or disliked.
>
> What is known must be appraised in its effects upon us before we can feel emotion.
>
> Our appraisal depends on the memory of earlier experiences with this situation or one like it; hence on our earlier appraisals and expectations.
>
> The various emotions are the result of different appraisals. Fear, love, anger, each implies a different relationship to the object, a different emotional experience, and a different action (always in animals, often in human beings). Since bodily sensations are different also, there must be differences in innervation to account for the differences in experience.

Whatever the brain mechanism mediating emotion, it must work in a way that will allow for the experience of emotion as we know it. It will be our task now to examine later neurological explanations to see whether the neural structures or circuits proposed conform to psychological requirements.

Papez's proposed mechanism of emotion. Papez's theory, first formulated in 1937, is a clear departure from Cannon's thalamic theory. Papez insisted that there must be definite cortical centers of emotion, though these should not be thought of as a "seat" of emotion. In his usage, they are simply the main stations in a complex dynamic chain of relays. To Papez it seemed equally unfortunate to insist on a strict cortical localization of function in the older sense, and to repudiate any and all notions of a central organization of nervous functions. Likewise, to disregard thalamic centers and pathways is as unjustified as to concentrate on them exclusively.

Since his is the first theory that suggests a definite brain mechanism activated during emotion, it deserves careful discussion. Curiously, psychologists have paid but scant attention to it—perhaps because it contradicts Cannon's theory but does so without any clear statement why an excitatory theory of emotion is preferable to a theory that explains emotion as release from cortical inhibition.

In his first publication (1937) Papez suggested that the stream of sensory impulses from the periphery takes three main routes from the thalamus to the cortex: one is the "stream of thought," going through the internal capsule to the lateral cerebral cortex; the second the "stream of movement," from the dorsal thalamus to the internal capsule, corpus striatum, and frontal lobe; the third is the "stream of feeling," from the central thalamus to the hypothalamus, and via the mammillary body and the anterior thalamic nucleus to the cingulate gyrus. In a later article (1939) Papez described these three afferent systems more extensively. The "stream of thought" was now called the "stream of perceiving." The "stream of movement" became a stream of excitations connecting the frontal lobes with the temporal and parietal lobes through the dorsomedial thalamic nucleus, thus serving "the global organization of the processes of thinking and comprehending." The "stream of feeling" became the stream of excitations that "underlies general consciousness and the emotive reactions."

However, Papez emphasized that it is only the combination of these three streams of excitations that yields psychological experience as we know it:

> The three receptive regions of the cortex, the posterior or sensory areas, the frontal lobe, and the gyrus cinguli, may be the recipients of these differently organized excitation streams which normally irradiate these regions of the cortex. It is also to be noted that these regions surround the motor and premotor areas which are located in their midst. If we construe these three incoming streams as representing the sensory, the global, and the emotive processes, it becomes evident that these must be combined in the cortex to yield integral psychological products. . . . That the cerebrum acts as a whole is a doctrine which does not repudiate the systematic and orderly organization of afferent and efferent impulses, but lays greater emphasis on their central organization. (1939, p. 156)

It appears from Papez's exposition that emotional experience can be aroused by incoming sensory impulses relayed via the subthalamus, mammillary peduncle, and mammillary body to the anterior thalamic nucleus and cingulate gyrus. Emotional experience can also be aroused from the cortex, by excitation either of the frontal lobes (via the medial forebrain bundle) or of the hippocampal gyrus (via the hippocampus-fornix system). In either case impulses will arrive in the mammillary bodies and be relayed to the cingulate gyrus which Papez considered the "receptive region for the experiencing of emotion." Emotional expression, according to Papez, is mediated by descending hypothalamic tracts in the mammillotegmental fasciculus which enters the brain stem and connects with the sympathetic motor neurons of the spinal cord; there is another connection with the tuber nuclei in the hypothalamus from which fibers descend to the hindbrain to excite parasympathetic nerves.

Papez's great contribution lies in postulating a definite area in the brain which mediates the experience of emotion. In addition, he sketched possible circuits connecting with this area. Instead of Cannon's notion that the thalamus adds an "emotional quale," Papez provided for a cortical region which mediates the experience of emotion, either on the basis of simple sensation or on the basis of a complex psychological experience for which he suggested a mediating mechanism (the stream of excitations connecting frontal, temporal, and parietal lobes).

There is still the difficulty of accounting for the conditions under which impulses will be relayed from the sensory system to the cingulate gyrus to be experienced as feeling or emotion. If every sensory impulse is relayed to hypothalamus and cingulate gyrus, it is difficult to explain why only some and not all sensations have an affective quality. In Cannon's theory, this difficulty is avoided by his assumption that a given thalamic pattern must be disinhibited before emotion can be experienced. The same difficulty in another form recurs when we turn to Papez's views on the *expression* of emotion. Emotional expression may be aroused either by cortical excitation or directly by the incoming stream of sensory impulses arriving in the hypothalamus. If induced by the hypothalamus, the expressive pattern could be initiated before the sensory impulses have arrived in the cingulate gyrus; that is, before the stimulus could be either perceived or appraised.

Psychologically speaking, an object must be known in some way before man or animal can react to it either with emotion or with action. Emotional expression that starts before a thing is perceived and is either liked or disliked is a logical impossibility, on Papez's own premises. Of course, reflexes sometimes occur without awareness of the object that aroused the reflex (e.g., a scratch reflex in a decorticated animal or the pupillary reflex to light during anesthesia). But whenever emotion occurs with the reflex there must be some knowledge of what is going on. When a man or an animal becomes annoyed at having a light directed into his eyes, there is no doubt that he is awake and experiences the annoyance. Perhaps some perception of the object or some experience of emotion is possible without cortex, but Papez's system does not seem to allow such an assumption. For him, the emotional experience occurs when hypothalamic impulses reach the cingulate cortex and are integrated with impulses from other cortical areas.

Despite these difficulties, Papez's theory is a milestone on the road. He suggested a definite neural circuit mediating the experience of emotion when impulses arrive in a particular cortical area. His theory makes it possible to explain the expression of emotion as a centrally initiated process, even though he also held that emotional expression can be aroused by sensory impulses relayed to the hypothalamus and cingulate gyrus on their way to the sensory cortex.

Papez supported his theory by quoting some neurological and clinical evidence. He mentioned that the essential damage in rabies has been found mainly in the hippocampus and the cerebellum; and rabies is characterized first by insomnia, irritability, and restlessness, which then usher in the stage of excitement and profound agitation. According to Papez:

> There is extreme hyperaesthesia to all forms of stimuli, such as light and sound, and every stimulus situation provokes great apprehensiveness and paroxysms of fear. The patient presents the appearance of intense fright and of mingled terror and rage. Hydrophobia, or fear of water, develops as a result of the painful pharyngeal spasm when the patient attempts to swallow fluids. Foaming at the mouth is the common symptom of autonomic disturbance. The predominance of hypothalamic lesions in the supraoptic nucleus has been described by Nikolau and Kopciowska. (1937, p. 733)

As evidence for the participation of the cingulate gyrus in emotion, Papez quoted reports of the mental state of patients with a tumor of the corpus callosum that encroached upon the gyrus cinguli. These reports mention apathy, drowsiness, indifference, loss of memory, disorientation in time and place, change in personality, "Witzelsucht," puerility; in a few cases, euphoria and depression. Since in all these cases the frontal and often the parietal lobes were damaged in addition to the corpus callosum and the cingulate gyrus, it is difficult to say which psychological changes represent the effects of cingulate gyrus damage.

Papez also mentioned that the precuneus, which broadens out from the cuneus on the medial surface of the cerebrum (see Fig. 1), is larger in males than in females and suggested that the precuneus may be the cortical area for sexual feelings, and thus a part of the emotional mechanism. Erickson (1945) later reported that a woman patient who had a tumor in the right paracentral lobule (connected with the precuneus) complained of insistent sexual sensations for more than ten years, which subsided as soon as the tumor was removed. This report seems to give substance to Papez's suggestion, though it is unclear just how such "sexual" sensations or feelings come about.

The evidence certainly suggests (though it does not demonstrate) that the hippocampus and cingulate gyrus play a role in emotion. Unfortunately, it does not give us reliable clues as to what that role could be. The reports of emotional changes after damage to the cingulate gyrus are difficult to assess and still more difficult to ascribe to specific lesions. It is not possible, either, to judge whether the experience of specific emotions or that of all feelings and emotions is mediated by the cingulate gyrus. Euphoria, puerility, emotional negativism, irritability, and agitation are mentioned, but apathy and indifference are too. We would have to assume that increased emotionality (e.g., euphoria, irritability, agitation) is a sign of the irritative stage of the disease process, while apathy and indifference are the effects of tissue destruction.

There is little evidence that fear or anxiety are induced by an irritative lesion of the cingulate gyrus.* The fear of water, supposedly characteristic for hydrophobia, seems to be rather an aversion to drinking because swallowing has become painful. We have shown in the preceding volume that fear is a reaction to danger when danger is difficult to avoid (cf. Vol. I, Ch. 10). The danger of swallowing can easily be avoided by not drinking.

Fear and anxiety are decidedly reduced by damage to the frontal lobes or prefrontal lobotomy; it seems that the frontal lobes rather than the cingulate gyrus mediate or at least facilitate the experience of these emotions. There also seems to be a reduction in perseverance and interest after prefrontal lobotomy, which fits more easily into Papez's conception of frontal lobe function. However, any theory of emotion would have to take into account the emotional changes that occur after prefrontal lobotomy, whatever other functions are mediated by these structures.

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* Smith (1945) and Ward (1948) reported that destruction of the cingulate gyrus produced "tameness" in monkeys; but we shall see in Ch. 2 that this is not an absence of fear.

To sum up: Papez's proposed circuit (hippocampus-fornix-mammillary bodies-anterior thalamic nucleus-gyrus cinguli) does seem to be active in emotion. But it is not certain whether this circuit is specific for emotion. In Chapters 2 and 3 we propose to review recent evidence that will give us a clue to the nature of this circuit. Papez's account also leaves unexplained why there should be such a reduction of anxiety after prefrontal lobotomy.

MacLean's conception of the "visceral brain." MacLean (1949) followed up the implications of Papez's theory and reviewed additional evidence showing that the structures within the emotional circuit are connected with the autonomic nervous system and important for survival. He pointed out that animals low in the evolutionary scale have a large medial olfactory tract which connects with correlation centers for smell, taste, and sensations from mouth and viscera. For lower mammals, smell is important not only for locating food or mate but also for avoiding danger. Later in evolutionary development, the lateral olfactory tract increased in size as compared to the medial olfactory tract. The lateral olfactory tract relays impulses to the somatic correlation center in the pyriform lobe, the anterior hippocampal gyrus (see Fig. 1).

According to MacLean, the whole hippocampal area is a correlation center for the most diverse sensations, all of them important for survival. The hypothalamus, which is connected with the hippocampal area (also called the rhinencephalon), provides these impressions with emotional coloring and serves as the autonomic effector organ:

> There are indications that the phylogenetically old brain (classically known as the rhinencephalon and arbitrarily referred to in this paper as the "visceral brain") is largely concerned with visceral and emotional functions. This region of the brain appears to be so strategically situated as to be able to correlate every form of internal and external perception. In other words, the possibility exists in this region for bringing into association not only oral (smell, taste, mouth) and visceral sensations, but also impressions from the sex organs, body wall, eye and ear. And in contrast with the neopallium, the rhinencephalon has many and strong connections with the hypothalamus for discharging its impressions. (1949, p. 351)

Such widespread connections suggest that the hippocampal system is of the greatest functional importance. Though the evidence from comparative anatomy suggests that somatic connections become increasingly important in evolutionary development, MacLean thought that the rhinencephalon is primarily concerned with visceral functions and elemental drives because this area continues to influence autonomic functions. He called it the "visceral brain" to distinguish it from the neocortex which controls body musculature and serves intellectual functions, and suggested that this primitive brain may serve the functions of the id, primarily concerned with oral-anal drives. The hippocampal cortex has a simpler structure than any other cortical area, a structure which does not differ much from mouse to man. This led MacLean to suggest that the hippocampal system can deal with information only in a crude and summary way, is unable to analyze language as does the neocortex, yet may be capable of nonverbal symbolism.

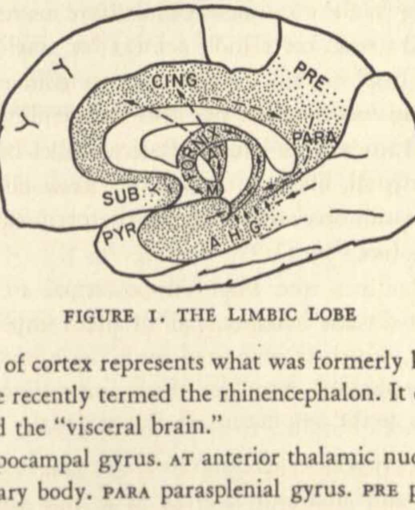

FIGURE I. THE LIMBIC LOBE

The shaded area of cortex represents what was formerly known as the limbic lobe of Broca, more recently termed the rhinencephalon. It corresponds to what MacLean has called the "visceral brain."

AHG anterior hippocampal gyrus. AT anterior thalamic nucleus. CING cingulate gyrus. M mammillary body. PARA parasplenial gyrus. PRE precuneus. PYR pyriform lobe. SUB subcallosal gyrus.

Source: P. D. MacLean. 1949. Psychosomatic disease and the "visceral brain": recent developments bearing on the Papez theory of emotion. Psychosom. Med. 11:338–53. Diagram specially redrawn.

If it is difficult to conceive how the hypothalamus, a relay station between periphery and cortex, could add emotional coloring to sensory impulses, it is still more difficult to imagine how the hippocampus, also a relay station (or "correlation center"), could be capable of nonverbal sym-

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bolism. Nonverbal symbolism, like emotion, implies a complex psychological experience; but of the two activities, that of associating, say, the color red "symbolically with such diverse things as blood, fainting, fighting, flowers" (1949, p. 348), is vastly more complicated. We have no evidence of nonverbal symbolism in animals, yet animals should be at no disadvantage if the hippocampus is an "animalistic and illiterate brain," as MacLean suggested.

MacLean further speculated that this primitive structure, old in evolutionary development, may in certain cases fail to establish normal connections with the sensorimotor neocortex: "In the psychosomatic patient it would almost seem there was little direct exchange between the visceral brain and the word brain, and that emotional feelings built up in the hippocampal formation, instead of being relayed to the intellect for evaluation, found immediate expression through autonomic centers" (1949, p. 350). Such speculation may be intriguing but should not be taken too seriously. The very fact that the hippocampus collects nerve impulses from all the sensory cortical areas surely indicates that it may integrate impulses relayed from the neocortex but cannot become autonomous in building up emotion.

Altogether, MacLean's stimulating article served to draw the attention of many workers in the fields of neurology, neurophysiology, and psychiatry to that mysterious part of the brain which includes the hippocampal system and the cingulate gyrus. The evidence he brought together showed this central region to be a most important correlation center for impulses from the neocortex. Later research has amply repaid the interest thus aroused.

Hebb's theory of emotion. The mechanism of emotion may also be explained as a sequence of brain activities regardless of their location. Earlier neurophysiological theories of emotion had assumed that emotion is a subjective experience mediated by neural circuits, even though theorists disagreed as to the nature of this experience: for James, emotion was a sensation complex, for Cannon, Papez, and MacLean, an experience in its own right. Hebb, however, does not acknowledge that emotion refers to any kind of experience. He defines it as "the neural process that is inferred from and causes emotional behavior, with no reference to consciousness" (1949, p. 148).

Hebb believes that explanations may be either psychological or neurological but not both; he chooses the more basic neurological explanation:

> Though it has been tried before now, an explanation cannot be half neural anatomy and half consciousness. They are different levels in a logical hierarchy. A problem recognized at one level can be resolved by recourse to a more fundamental set of conceptions, but an hypothesis cannot well comprise two levels, take in two universes of discourse at the same time. (1949, p. 3)

One may well ask why an explanation may not take in two levels, two "universes of discourse"? Our knowledge is not restricted to one level: we know what we experience, whether such experience is emotional or cognitive. The scientist is free to take into account his own experience when he receives good or bad news and compare his reaction with that of another whom he observes as that one receives good or bad news. He is also free to observe muscles and nerves, brain structures and circuits under various conditions, both in man and animal, in the living or in the dead body. The behaviorist may arbitrarily restrict himself to one kind of experience, namely observing the behavior of others. And he may stipulate, either that it must be explained exclusively on the behavioral level, or exclusively on the neural level; but such restrictions are not inherent in the scientific enterprise. As long as nerves and muscles are active while we have an experience and act upon it, men may legitimately ask what structures are active in a given experience or action. If the psychologist's misguided purism prevents him from investigating certain topics within his own field, scientists from other disciplines will have to fill the gap. Hence we see physiologists and neurologists propounding theories of emotion (see Cannon, Papez, MacLean) while psychologists keep scornfully aloof. Or we find neurologists and anatomists wrestling with the problem of consciousness while psychologists consider it merely a semantic pseudo-problem (see Lashley, Hebb, in: Delafresnaye, ed. 1954).

When it comes to proposing a strictly neurological explanation for psychological activities, we stumble against the cold fact that such an explanation is of no use because it does not apply to human experience. At best, neurological explanations must be translated into psychological terms before their implications can be grasped—and this is precisely what Hebb has done: his strictly neurological discussion is interspersed with psychological digressions so that it almost seems as if two separate books were combined into one. At worst, in a neurological explanation terms are used in such a way that they refer to psychological rather than to neural activities: nerves and brain cells (or muscles) are made to do what the person does. This is a predicament familiar to behaviorists, and Hebb does not escape it. For better or worse, the psychological explanation is obligatory, though by itself not sufficient. While psychological activities cannot be reduced to neural processes, our psychological understanding will not be complete unless we know the neural pathways that mediate our experience.

Hebb chooses a neurological theory in preference to a psychological explanation because "writers on the topic of emotion insist that there is no characteristic conscious event that can be called an emotion" (1946, p. 101). The writers he quotes (Dashiell, Dunlap, Harlow and Stagner, Landis, Lashley, Young) are either neobehaviorists who avoid conscious states by conviction or psychologists who deny that emotion has any separate status at all (Duffy, 1934). Yet emotions are facts of common experience. Even an objective psychologist like Knight Dunlap says: "When the plain man speaks of fear, rage or grief, he apparently has reference to some facts—moreover, to facts which are, or can be experienced (and experience is an occurrence, and an undisputed fact)" (1928, p. 151).

Now experience is something lived through *of which we are aware.* Hebb recognizes that awareness is part of emotion:

> Psychologists agree that there is no special conscious quale of an emotion; but this does not imply that there is no *awareness of a tendency toward a certain kind of action,* any more than the experimental failure to find specific patterns of muscular action is a denial that a frightened person may run away, or that an angry one may strike. . . . How such awarenesses are to be explained psychologically I do not pretend to know; but it is clear that they exist. (1946a, p. 103; italics added)

If the awareness of a tendency to a specific action exists in emotion, need we look further for a "special conscious quale" of the emotion?

The psychologists quoted by Hebb also insisted that there is no recognizable pattern of emotional expression. Yet Hebb found that the emotions of chimpanzees can be recognized and their actions predicted if "frankly anthropomorphic concepts of emotion" are used, while an objective description of their behavior resulted in an "almost endless series of specific acts in which no order or meaning could be found" (1945a, p. 88). Such recognition of an animal's emotion is immediate, "intuitive," according to Hebb, and thus cannot be based on "the inference that one of these postulated (neurological) states exists in the subject" as he later insisted (1949, p. 148).

One of the caretakers in the colony, for instance, said of the chimpanzee

Bimba that she is "quick to resent slights," that she "gets mad over nothing at all," observations Hebb found apt and accurate. Such remarks surely are the fruit of the man's own experience of slights and irritations, not the result of an inferred neural state that is quite unknown to him. The only reason why anthropomorphic terms make it possible to recognize emotions is the fact that they refer to some experience which is recognized in other human beings and even in animals. If an observer is able to recognize a given behavior pattern as belonging to one and the same emotion in spite of individual variations and in spite of variations in different situations, he must do it by referring certain responses to something he knows which unifies them into a variable pattern. He cannot refer these responses to a hypothetical neural state because he does not know that state nor does he know what patterns such a state could induce. If it were possible to work out an emotional pattern by reasoned inferences, it could have been done from the careful objective descriptions of the chimpanzee's behavior.

When Hebb refuses to define emotion in experiential terms, he turns his back on concepts that had served him well for the recognition of the phenomenon—in favor of a hypothetical neural state which could never contribute to either recognition or description of emotion. Hypothetical constructs and intervening variables are useful and sometimes indispensable when they refer to a process that cannot be observed. But to substitute such constructs for an experiential fact is of doubtful value.

But let us see how Hebb describes the neural processes which he equates with psychological activities:

> Any frequently repeated, particular stimulation will lead to the slow development of a "cell-assembly," a diffuse structure comprising cells in the cortex and diencephalon (and also, perhaps, in the basal ganglia of the cerebrum), capable of acting briefly as a closed system, delivering facilitation to other such systems and usually having a specific motor facilitation. A series of such events constitutes a "phase sequence"—the thought process. Each assembly action may be aroused by a preceding assembly, by a sensory event, or—normally—by both. The central facilitation from one of these activities on the next is the prototype of "attention."
>
> It is proposed also that there is an alternate, "intrinsic" organization, occurring in sleep and in infancy, which consists of hypersynchrony in the firing of cortical cells. But besides these two forms of cortical organization *there may be disorganization*. It is assumed that the assembly depends completely on a very delicate timing which might be disturbed by metabolic changes as well as by sensory events that do not accord with the pre-existent central process. When

> this is transient, it is called *emotional disturbance;* when chronic, neurosis or psychosis. (1949, p. xix; italics added)

Thus emotional disturbance is cortical disorganization of phase sequences organized into cell assemblies. The phase sequence (which may be a perception, a thought, an image, or a concept) is fleeting and continually needs new sensory content to maintain its organization. According to Hebb:

> Some phase sequences are more firmly established than others: that is, more capable of perpetuating themselves. The level of stimulation from sense organs affects the persistence, and a phase sequence may perpetuate itself by raising this level, as when one knits his brow and pinches himself to stay awake. (1949, p. 222)

But to knit his brow and pinch himself, a man must decide to stay awake rather than go to sleep. If staying awake is so difficult that he has to pinch himself, the phase sequence must be very unstable. Before it can be stabilized, another chain of phase sequences must be established (the decision to stay awake and pinch himself), which can hardly be done by the unstable sequence. Pinching or knitting one's brow seems to be the sensory content which stabilizes an unstable phase sequence; but in that case, not only the stimulation but the decision to apply it must come from somewhere (but where?) outside that sequence.

A phase sequence, according to Hebb, can maintain itself only when it continues to develop, "to lead to new combinations of assemblies, which in turn means new perceptions, new insights, new ideas" (1949, p. 233). Hence "one would find behavior dominated always by the thought process that is not fully organized—one that is achieving a new organization or one in which synaptic decay makes it necessary that organization be reachieved" (1949, p. 229). But is it really the phase sequence that achieves or reachieves organization? Here is the ambiguity at the root of Hebb's system. He equates "phase sequence" with thinking or perceiving and then takes the result of such psychological activity to be the product of neural activity. If it were really the phase sequence which organizes and perpetuates itself, there could be no difference among a chain of associations, a daydream, or a closely reasoned train of thought. Whatever the thought content, the phase sequence could not recognize it. Surely, neither the phase sequences already established nor incidental sensory stimulation can determine what the new organization is going to be. No doubt, phase sequences are active when a given organization of thought is achieved, but they do not create it. It is the thinker who either indulges in idle dreaming or disciplines his thought toward a logical conclusion.

According to Hebb, sensory stimulation not only provides new content for phase sequences, but may also disturb the existing organization. Such disturbance results in the conflict emotions. This is how Hebb explains fear:

> The immediate source of fear is a disruption of a coordination, principally acquired, in the timing of cellular activities in the cerebrum. The disruption may be due to conflict, sensory deficit or constitutional change. With disruption there at once occur processes tending to restore the integration of cerebral activities; in fear these include either liminal or subliminal activation of processes determining avoidance. Optimally avoidance tends toward completely averting the cerebral disruption, and at this stage avoidance without fear would be said to occur. (1946b, p. 271)

If emotional disturbance is a disruption of cerebral organization, pleasant emotions are its continued growth and development:

> Pleasure is not the activity of particular structures in the nervous system, and not even a particular kind of pattern of cerebral organization. It is thus necessarily a transient state of affairs in which a conflict is being reduced, an incipient organization being dissipated, or a new synthesis in assembly action being achieved. (1949, p. 232)

Thus emotions are either developing phase sequences or disrupted phase sequences; the former are pleasant, the latter unpleasant emotions.

This is the same analogical thinking we have found in Hebb's conception of neural organization. Throughout, his explanations depend on concealed psychological causation. To say that incoming sensations can disrupt a phase sequence is to assume that each phase sequence must be different because sensations and thoughts are different. But by definition, the only difference between phase sequences is a difference in the frequency of the nerve impulses forming the circuit. Now we know that increasing intensity of stimulation will increase the frequency of nerve impulses. But is there any ground for assuming that an unpleasant surprise must necessarily induce a frequency incompatible with existing frequencies, while a pleasant surprise will always induce compatible frequencies? To suppose that an emotional disturbance must be a disruption in the timing of the nerve impulses that mediate it is like saying that the report of a disaster has aroused consternation and feverish activity in the hearers because it was jumbled in transit. If the message had been jumbled, it would not be understood and would leave the hearers indifferent. If phase sequences were disrupted in transit, they could not organize the behavior patterns we recognize as pain or fear or rage.

In later publications, Hebb explained that the terms he uses are really psychological constructs:

> The "phase sequence" is a psychological rather than a physiological conception. Its chief specifications were determined in an effort to account for certain facts of behavior. Further, the "cell assembly" is more a behavioral than a physiological conception; it was the behavioral data that led to the general idea in the first place, and I had to put some strain on the physiological and anatomical data to make it intelligible in these terms. This then is a case of taking two psychological conceptions, the train of thought and the idea (or image), and attempting to give them neurophysiological specifications. (1958, p. 461)

When we ask what Hebb means by a psychological construct, he answers:

> What I mean by a psychological as opposed to a physiological construct, therefore, is that its referents are primarily in the behavior of the intact animal. One may name it, and hypothetically describe it, in physiological terms; but this is in the effort to maintain communication between different levels or universes of discourse. (1959, p. 632)

Reading these and similar passages, we begin to wonder what language Hebb is using. He calls "phase sequences" and "cell assemblies" psychological conceptions when the terms properly apply to the realm of physiology. In like manner, he passes freely from one universe of discourse to another, using terms in both universes that properly belong to only one of them. Such a procedure can be understood only on the basis of a *simpliste mystique* concerning the structure of the world: in Hebb's world, there seem to be levels of reality from molecular to molar, where little hunks of stuff can be heaped into bigger and bigger hunks, depending on the way in which one is thinking about them. The subatomic world *is* real (and no other is at the moment) whenever we think of electrons, protons, etc., and is *real* only because we think of it. Since this kind of *thinking* and *making real* or *being real* is supposed to be all the same thing, language proper to thinking may be applied indiscriminately to *being*, on the same and on a different level of aggregation. The world can be chopped into smaller bits and pieces and, by the same token, so can thought. I can think "chair" (one level of aggregation, real in its own way), and, on a lower level, call my *thinking*, lignin and wood fibers. This seems to be the mystique of Hebb's theory. Even if we seem to be reading into Hebb's discourse a dialectic which he would repudiate, it is only on this basis that his system becomes consistent. This becomes particularly clear in the light of the clarification of his position given in the two articles from which we quote.

Concerning emotion, Hebb admits in his most recent paper:

> On emotion, I gave the impression that the disruption of assembly action can be deduced from the physiological and anatomical postulates I had made. . . . This deduction . . . does not stand up on subsequent analysis . . . and I must regard this instead as an inference from behavior, not well justified physiologically. I am inclined now to think more in terms of some sort of disorganization of the phase sequence, involving overfacilitation from the arousal system rather than of the individual assembly, but this at best remains quite vague. (1959, p. 639)

It should be added that Hebb's other theoretical conceptions also cannot be deduced from his physiological and anatomical postulates. In all fairness, however, it ought to be said that many important neurological discoveries, for instance, the arousal system to which he refers in the above quotation, had not been reported at the time Hebb formulated his theory. We propose to review the newer neurophysiological research findings in the next four chapters and hope to show that connections in the brain mediating various psychological activities—from perception to memory, emotion, and action—can be identified.

2. FUNCTIONAL SEQUENCE: I. APPRAISAL

In our discussion of Papez's theory and the later contribution of MacLean we noted that an emotion can occur only after the situation is perceived and appraised. This requires that different sensory impulses arrive in the cortex and be integrated and analyzed before the mechanism serving emotional experience and expression can go into action. Vision, hearing, touch, taste, or smell merely inform us that there are colors, sounds, feels, tastes, scents. Some integrating system is needed to transform these isolated sensations into an object that has a certain shape, sound, feel, taste, or odor. However, neither single sense impressions nor their integrated functioning which provides us with objects can inform us of the effect of this particular object on us, or advise us to approach or avoid it. Some functioning system is required that will use the integrated product of sense impressions, will mediate some awareness of its import, and will provide access to somatic and autonomic motor systems that carry out appropriate action, emotional expression, and autonomic changes.

This and the next two chapters will be devoted to an attempt to trace the circuits that are active during the whole sequence from perception to appraisal and the felt emotional impulse that leads to appropriate action. In recent years a great deal of research has accumulated, which we shall try to examine, sift, and integrate. In this process some currently held opinions may have to be revised and some accepted hypotheses may have to be extended. Since research activity in this field is lively, our integration cannot be final and may eventually prove wrong in many details. At the same time, the attempt to map out the sequence of neural events in emotion in as much detail as possible should prove fruitful because details can be checked, proved, or disproved, while general models and theories, though brilliant and stimulating, may be in the end a barrier to knowledge because they are based on analogies instead of verifiable facts.

There seems to be general agreement that the limbic system is involved in emotion and, particularly, that the hippocampus receives nerve impulses from various cortical areas and relays them to the subcortex (cf. Papez, 1937; MacLean, 1949; Fulton, 1951a; Kaada, 1951, 1953; and others). But so far, there has been no suggestion as to just how sensory impulses are integrated and transformed into emotion or even what is the actual function of the limbic system in this integration.

STRUCTURE OF THE LIMBIC SYSTEM

Since the limbic system seems to be involved in emotion, let us first take a look at this system and its connections. Broca, who first coined the term "limbic lobe," explained that the name indicates the region that surrounds the hilus of the hemisphere (*le limbe de l'hémisphère*). He included in it the olfactory tubercle (anterior perforated substance in man); the hippocampus and hippocampal rudiment arching from the septal area over the corpus callosum and ending in the hippocampus; the cortex near the olfactory striae and the prepyriform area (uncus in man); the hippocampal, parasplenial, cingulate, and subcallosal gyrus (Fig. 1). There are additional regions, i.e., the frontotemporal cortex, a junctional band of tissue between the prepyriform area and the orbitofrontal as well as the anterior temporal neocortex, which are interconnected and have the same cell structure, and have been assigned to the limbic system. Neuronographic * studies by Pribram and MacLean (1953) have shown that six different regions can be identified in the limbic system which are connected with different extralimbic cortical areas (Fig. 2).

Developmentally, the limbic lobe includes the most primitive type of cortex (called entopallium, archipallium, or allocortex) and a transitional type (mesopallium, paleo- or juxtallocortex). The entopallium includes the olfactory bulb and tubercle (anterior perforated substance in primates), the area of the diagonal band of Broca, septal area, prepyriform cortex (i.e., the area surrounding the lateral olfactory tract), periamygdaloid cortex (i.e., the corticomedial division of the amygdala), and the hippocampus with dentate gyrus and hippocampal rudiment. The mesopallium includes the cingulate, retrosplenial, and hippocampal gyri, the island of Reil and operculum, and the subcallosal and frontotemporal transitional cortex (see Pribram and Kruger, 1954).

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* Neuronography is a method developed by Dusser de Barenne and McCulloch (1938) in which parts of the brain are painted with strychnine solution. The evoked potentials are then recorded by needle electrodes inserted in various brain regions. The areas "fired" by strychnine are projection areas of the stimulated region.

Structurally, the entopallium is the simplest type of cortex. It consists essentially of three layers. The superficial (supragranular) layer is poorly developed and the afferent plexus lies immediately below it. The receiving cells in the next layer are intermingled with the pyramidal cells which give rise to motor impulses. The mesopallium is more highly developed; in it the receiving (granular) layer is completely separate from and superimposed upon the pyramidal layer. As the mesopallium shades into the ectopallium (neocortex), the layers gradually become separated and specialized until six layers can be distinguished. In the ectopallium the cells receiving the afferent plexus are in the fourth (internal granular) layer, while the efferent fibers arise from the fifth (pyramidal) layer (Fulton, 1951b).

It used to be thought that the hippocampus represents part of the "olfactory brain" or "rhinencephalon." In the last two decades, stimulation studies have made it clear that there is no direct connection between the olfactory tract and the hippocampus. Brodal (1947) reviewed the wealth of evidence in detail and concluded that this structure is not part of the olfactory system. Rather, the hippocampus (including the hippocampal rudiment), receives impulses from every cortical area (MacLean, 1949). Hence we propose to distinguish the *hippocampal system,* in which we include the hippocampal rudiment and hippocampus together with the subiculum and the *fascia dentata.* The hippocampal rudiment, arching from the septal area over the corpus callosum to connect with the hippocampus, is an attenuated form of this structure. In the foetal brain, the hippocampal structure with dentate gyrus and subiculum is still recognizable in the hippocampal rudiment (Hines, 1922). We also propose to distinguish the *limbic system,* composed mainly of transitional cortex (as compared with the three-layered entopallial cortex of the hippocampal system). The limbic system includes the subcallosal, cingulate, retrosplenial and hippocampal gyri (entorhinal cortex and adjacent presubiculum), and the island of Reil. This is a functional division and will be explained later in this chapter.

From the hippocampus, an efferent tract, the fornix, sweeps in several divisions forward and downward to the hypothalamus. The precommissural part goes to all septal cell groups and to the preoptic hypothalamic area. The postcommissural fornix runs to the dorsal hypothalamic area and periventricular hypothalamic zone, to the tuber region and the mammillary body. There is a massive direct projection to the anterior thalamic nucleus and the medial thalamic region (Simpson, 1952; Powell and Cowan, 1955; Nauta, 1956). The mammillary bodies in the hypothalamus are connected via the mammillothalamic tract with the anterior thalamic nucleus and the cingulate gyrus, and via the mammillotegmental tract with the brain stem tegmentum and reticular formation. The brain stem reticular formation is connected with the primary sensory pathways and sensory cortical areas (cf. French *et al.*, 1953).

MECHANISM SERVING THE INTUITIVE ESTIMATE

We know that sensory experience is specified by the receiving area, not by the kind of stimuli applied. A visual sensation is experienced when the lateral geniculate body in the thalamus and finally the striate area in the occipital cortex are excited, whether that is done on seeing an object or on electrical, mechanical, or chemical stimulation of visual nerves or visual cortical areas. Sensory experience seems to be possible even after the removal of the cortical receiving areas, provided the sensory thalamic nuclei are intact. But this is merely the experience of crude sensation, without pattern, color, or spatial dimension. Since emotion is an experience *sui generis,* as specific as sensation though based on it (see Vol. I, Ch. 9), there should be, analogous to sensation, a cortical as well as a thalamic region to mediate such an experience.

We can like or dislike only something we know. We must see or hear or touch something, remember having done so or imagine it, before we can decide that it is good or bad for us. Sensation must be completed by some form of appraisal before it can lead to action. Most things can be evaluated only when they are compared with similar things in the past and their effect on us. What is sensed must be appraised in its context, in the light of experience; accordingly, our evaluation in many cases will have to draw upon memory.

Appraisal seems to be a process of evaluating and comparing sense impressions and memories from many sense modalities, and thus may take an appreciable time. But once a thing is evaluated, the experience of liking or disliking it follows immediately and automatically. It is a momentary experience, as fleeting as sensation, even though it may endure if the process of appraisal continues, or may even change as different aspects of the situation are assessed. Appraisal is a unitary function of which we may be aware when it is prolonged (e.g., in deciding that we like the taste of a new drink), but of which we may also be completely unaware when it is immediately followed by emotion (e.g., on seeing a snake and getting scared).

Since appraisal and emotion complete sense perception, it would seem likely that the process of evaluation is mediated by a circuit that relays sensory impulses to an area which mediates liking and disliking, pleasure and pain. We know that animals can react to intense sensations (e.g., a pinch) with pain even after destruction of the somesthetic cortical area. Thus a rudimentary evaluation of sensory stimuli seems to be mediated by subcortical relays, and the resulting experience of liking or disliking, by some thalamic area.

We hope to show that this appraisal and the following experience of liking or disliking are mediated by a special system that receives relays from the sensory system. It includes the afferent connection from sensory receptors to the brain stem reticular formation, the intralaminar and midline thalamic nuclei, and the cortex of the limbic lobe. We propose to call this system of relays the *estimative system* * because it serves the evaluation of incoming sensations. This estimate becomes progressively more accurate as the relays reach the thalamus and limbic cortex. Even without the cortex, the animal is apparently able to estimate the intensity of sensations, as long as the medial thalamus is intact. Without the somesthetic cortex, man or animal can still experience pleasure or pain but exact localization is now impossible.

There is evidence that makes such a suggestion plausible. The nuclei of the midline and the intralaminar thalamic nuclei receive fibers from the spinothalamic and trigeminothalamic tracts and from the medial lemniscus, both directly and via the reticular formation in the medulla. These nuclei are among the phylogenetically oldest in the thalamus and mediate a limited appreciation of pain (Walker, 1938). So-called "thalamic pain" (which is diffuse and poorly localized) persists when the somesthetic cortical area is damaged and even when the somesthetic thalamic nuclei are destroyed. It disappears when the centrum medianum nucleus (one of the intralaminar thalamic nuclei which receives relays from the somesthetic projection of the medial lemniscus and the brain stem reticular formation) is also destroyed (Hécaen *et al.*, 1949; Bowsher, 1957; Bishop, 1959). When sensory impressions are received and registered in sensory cortical areas without the possibility of appraising them, we would expect that any "liking" or "interest" in what goes on would be missing and that the individual would be completely out of touch with the environment. This is actually what happens. Destruction of the medial brain stem or the medial thalamus (our subcortical estimative system) leads to akinetic mutism, coma, or death, depending on the extent of the lesion (Bailey and Davis, 1945; Jasper, 1955).

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* To call the whole system the "limbic system" would be inappropriate because the only "border" Broca referred to is the inner border of the hemisphere. Since we propose a definite function for this system and will show later (pp. 38 ff., Ch. 3, pp. 94 f., Ch. 5, pp. 153 ff.) that this function is served by cerebral, spinal, and peripheral pathways, it seems preferable to indicate this function by an appropriate name. In the same way, the "sensory system" and the "motor system" are systems that mediate particular functions.

In man, lesions in the neighborhood of the third ventricle (i.e., in the medial thalamus) have produced akinetic mutism. The patient lies with eyes open and muscles relaxed, neither speaks nor moves voluntarily, but may withdraw his hand when it is pricked (Cairns, 1952; Nielsen, 1951). Interestingly enough, Nielsen suggested that conation (the "desire to move") has a mesencephalic, a diencephalic, and a cortical component, and commented that the "fundamental defect in akinetic mutism is . . . one of emotion, the loss of which removes all desire to move" (p. 461). We will see later how emotion leads to action. Here it is sufficient to note that what we perceive must concern us, that we must like or dislike it before we want either to approach or avoid it. If we cannot establish such personal relations with what we see, hear, touch, smell, or taste, we do not bother even paying attention to it.

The appraisal of objects and of their effect on us (as contrasted with the evaluation of sheer sense impressions) seems to require a relay to the cortex. We would suggest that the experience of liking or disliking *some particular object* is mediated by the cortical limbic areas. These areas allow for an estimate of various qualities of the object and the direction in which it is to be found; they also allow for memory. The fact that all vertebrates have a limbic lobe very similar in structure seems to speak for the notion that the limbic system mediates an experience fundamentally similar in nature. All vertebrates show the ability to evaluate their environment and take appropriate action. They can assess "instinctively," on first encounter, what is good or bad for them (e.g., their natural food or mate). They are also able to act according to what has been good or bad for them in the past. The dog learns to greet his master's car joyously, the horse develops a liking for sugar, and the child can acquire a taste for beer, wine, or birds' nests, depending on whether he is brought up in Germany, France, or China.

That the cortical limbic regions receive relays from the thalamic midline and intralaminar nuclei is shown by the findings of Rose and Woolsey (1949) that these nuclei degenerate only when limbic lobe, hippocampus, amygdala, and striatum are destroyed in addition to the neocortex. We shall see later that the thalamic estimative system projects to all these areas. Since Pribram and McLean (1953) have found that the excitation of sensory, motor, and association areas produces potentials in each adjoining limbic region, it seems likely that different limbic areas mediate the experience of liking and disliking of the objects that are perceived via the corresponding sense modalities. Only in rare cases is the impression received via one sense modality sufficient for evaluation, for instance, when a sharp blow is felt as painful. Most of the time, something that is new in our experience must be touched as well as seen and heard, and sometimes even smelled or tasted, before we can decide whether it will benefit or harm us. The information from the various senses must be collected, compared, and assessed before we can like or dislike this particular thing. Accordingly, the various limbic regions must be interconnected with one another and must also be connected with specific sensory areas—which is precisely what has been found (cf. Pribram and MacLean, 1953; Pribram and Kruger, 1954).

Even before we can identify something we may like or dislike it. The infant immediately likes milk and dislikes vinegar. The adult immediately likes a harmonious chord, diffuse light, the green of trees; and dislikes a blinding light, glaring colors, a clashing discord. There seems to be an appraisal of the sensation itself, its quality and intensity, before the object is identified and appraised. The first fruit of perception, the first affective experience, seems to be a feeling rather than an emotion, following the appraisal of how this aspect of an object, perceived via a particular sensory avenue, affects me. All sense impressions are assessed first as to their effect on the sensory system. This preliminary appraisal seems to occur as neural impulses are relayed from the sensory receptors to the midline and intralaminar thalamic nuclei; it becomes the evaluation of something specific when these relays reach the limbic cortical region.

This first feeling ordinarily does not last long enough to stand out against the later appraisal of the object and its effects on us, which is followed by emotion. Only intense stimulation, taxing the sensory system, or near-threshold stimulation perceived with considerable effort, will be felt as painful and uncomfortable whatever the object that arouses it. Ordinarily, the mild pleasantness or unpleasantness of the sensation is swallowed up in the evaluation of the object as a whole. Our discussion in Chapter 4 of the preceding volume, in the section Attention and Feeling, has shown that feelings must normally be focused on, or attended to, before they will be noticed. The evaluation of the object as it affects us must be held in abeyance if we want to concentrate on its effect on us via one particular sense modality.*

We shall first discuss the experience of liking and disliking as mediated by various limbic areas, no matter whether it is an isolated feeling or the starting point of an emotion, no matter whether it is based upon the appraisal of a sense impression or the appraisal of an object. Then we shall examine the psychological process of evaluating an object, so as to isolate the neural connections that mediate it.

Of course, we hardly ever evaluate something as good or bad and like or dislike it without being urged to some action. It is either good to eat or to pet, to fight or to avoid. This impulse to action is just as much part of the complete emotion (e.g., desire, affection, anger, fear) as liking or disliking. Hence we have defined emotion as the "felt tendency toward something appraised as good (suitable) or away from something appraised as bad (unsuitable)" (see Vol. I, Ch. 9). Now it is unlikely that this impulse to action is mediated by the estimative system which, we believe, mediates liking or disliking. But we know that an impulse or desire for action is often experienced when the motor area is stimulated in a conscious patient (Bucy, 1944). And we might expect that such an urge to action will be aroused when something is appraised as good for a specific action. If this analysis is correct, neural impulses from the limbic areas would have to be relayed to the motor areas before we would experience an urge to action. The neural circuit serving action will be discussed after we have established the connections which mediate an appraisal and the following experience of liking or disliking.

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* This formulation agrees with Gemelli's notion that each emotion is preceded by a feeling (see Vol. I, Ch. 8), though we would say that this feeling is based on the intuitive appraisal of the quality or intensity of sense impressions. For this reason, it can only be a feeling of liking or disliking, pleasantness or unpleasantness, and not a feeling of strain, lightness, strength, etc. The attitude needed to report on feelings consists in deliberate attention to the way in which we are affected by a particular sense impression (cf. Vol. I, Ch. 4 and Beebe-Center, 1932).

THE EXPERIENCE OF LIKING/DISLIKING

According to Pribram and MacLean, there are five well-defined limbic areas in the limbic system which have a two-way connection with the sensory and association cortex (see Fig. 2). While all limbic areas are interconnected, each separate area has a two-way connection with only one neocortical region. Now we do not know whether these two-way connections within each region form a functional circuit. But we do know that the relay from sensory area to limbic region is functioning at least during sensory experience. Sensory stimulation is known to evoke potentials in the appropriate sensory cortical regions and also in the neighboring limbic regions. We can assume that such potentials indicate a psychological function that is intimately bound up with sensation and follows immediately upon it.

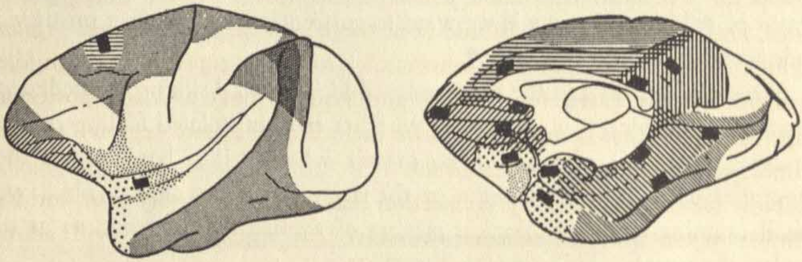

FIGURE 2. LATERAL AND MEDIAL SURFACE OF A MONKEY BRAIN, SHOWING THE DISTRIBUTION OF LIMBIC AND EXTRALIMBIC REGIONS

Coarse stipple or striation indicates regions with two-way connections as mapped by physiological neuronography. Finer stipple or striation shows additional cortex activated in one direction from a point within the region. Black rectangles show sites of strychninization.

Source: K. H. Pribram and P. D. MacLean. 1953. Neuronographic analysis of medial and basal cerebral cortex. II. Monkey. J. Neurophysiol. 16:324–40.

At the very least, these limbic regions represent a relay from sensory areas to adjoining limbic areas. The *medial occipitotemporal region,* for instance, connects the cuneus, fusiform, and lingual gyri (visual sensory and association area) with the limbic cortex of the posterior hippocampal gyrus. The *medial parieto-occipital region* connects the precuneus (somatosensory and association cortex) with the limbic cortex of the posterior cingulate and parasplenial gyri. The *medial frontoparietal* region embraces the superior frontal neocortex (motor and premotor region) and the limbic cortex of the anterior cingulate gyrus. The *medial frontal region* includes the ventromedial neocortex of the frontal lobe (including the olfactory area) and the limbic cortex of the subcallosal and medial frontal orbital surface. Next, the *medial frontotemporal* region connects the neocortex of the frontal pole and the ventromedial aspect of the anterior temporal pole with the limbic cortex of the limen insulae, prepyriform area, and anterior insula. In addition, there is a *medial temporal region* which represents a *one-way* connection from the superior and middle temporal gyrus (area for hearing and equilibrium) and the posterior operculum (somatosensory face area including the taste region) to the limbic cortex of the posterior insula.

Now we can examine the connection between each sensory area and its limbic region and see whether we have any evidence that will allow us to infer that sensory impressions are appraised in a rudimentary fashion in the medial thalamus, and objects in the limbic region nearest the sensory cortical area which mediates their perception.

Evaluation of equilibrium. The vestibulary cortical zone that mediates the sense of equilibrium seems to lie in the depth of the posterior lateral fissure, in the posterior part of Brodmann's area 22 (Walzl and Mountcastle, 1949). According to Pribram and MacLean, this area also is connected with the limbic cortex of the posterior insula. A sudden change of position may immediately be felt as uncomfortable, probably via the medial thalamus which receives fibers from the medial longitudinal fasciculus carrying vestibular impulses (Spiegel, 1934). An uncomfortable position is corrected via connections from medial thalamus to midbrain tegmentum and brain stem reticular system. An animal retains the righting reflex not only after decortication but even after the brain stem is transsected at the rostral level of the inferior colliculi (cf. Peele, 1954). This leaves a direct connection from the sensory pathway to midbrain tegmentum and brain stem intact. However, the proper balance in running, skating or skiing requires an exact appraisal of position which seems to be mediated by the cortical limbic region in the posterior insula (Figs. 3, 4).

Evaluation of sounds. The auditory area is on the upper surface of the

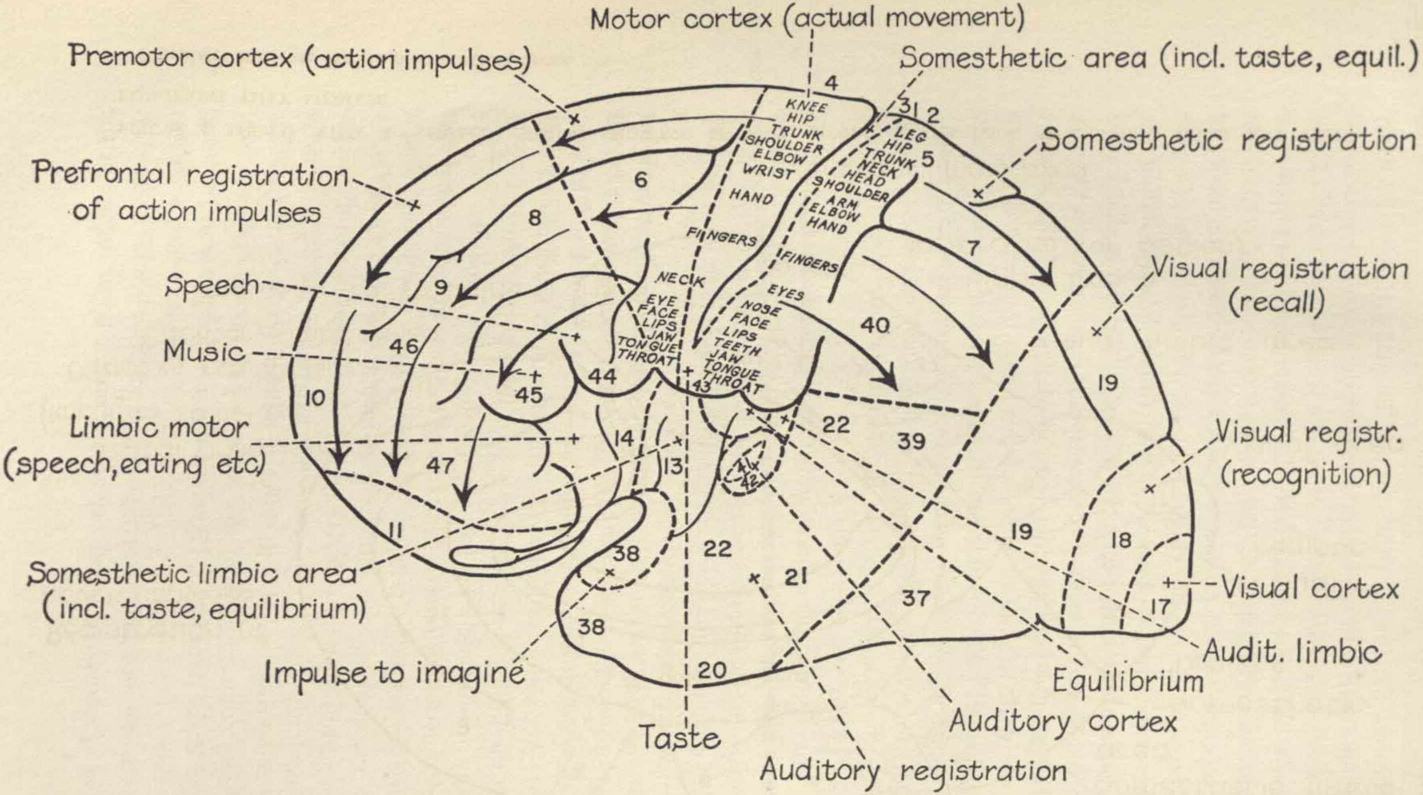

FIGURE 3. LATERAL VIEW OF HUMAN BRAIN WITH INSULA SPREAD OUT, SHOWING SENSORY, MOTOR, ASSOCIATION, AND LIMBIC AREAS, AND THE FUNCTIONS THEY MEDIATE

Arrows indicate that motor and somesthetic representation continues into the association area. Numbers refer to Brodmann cortical areas.

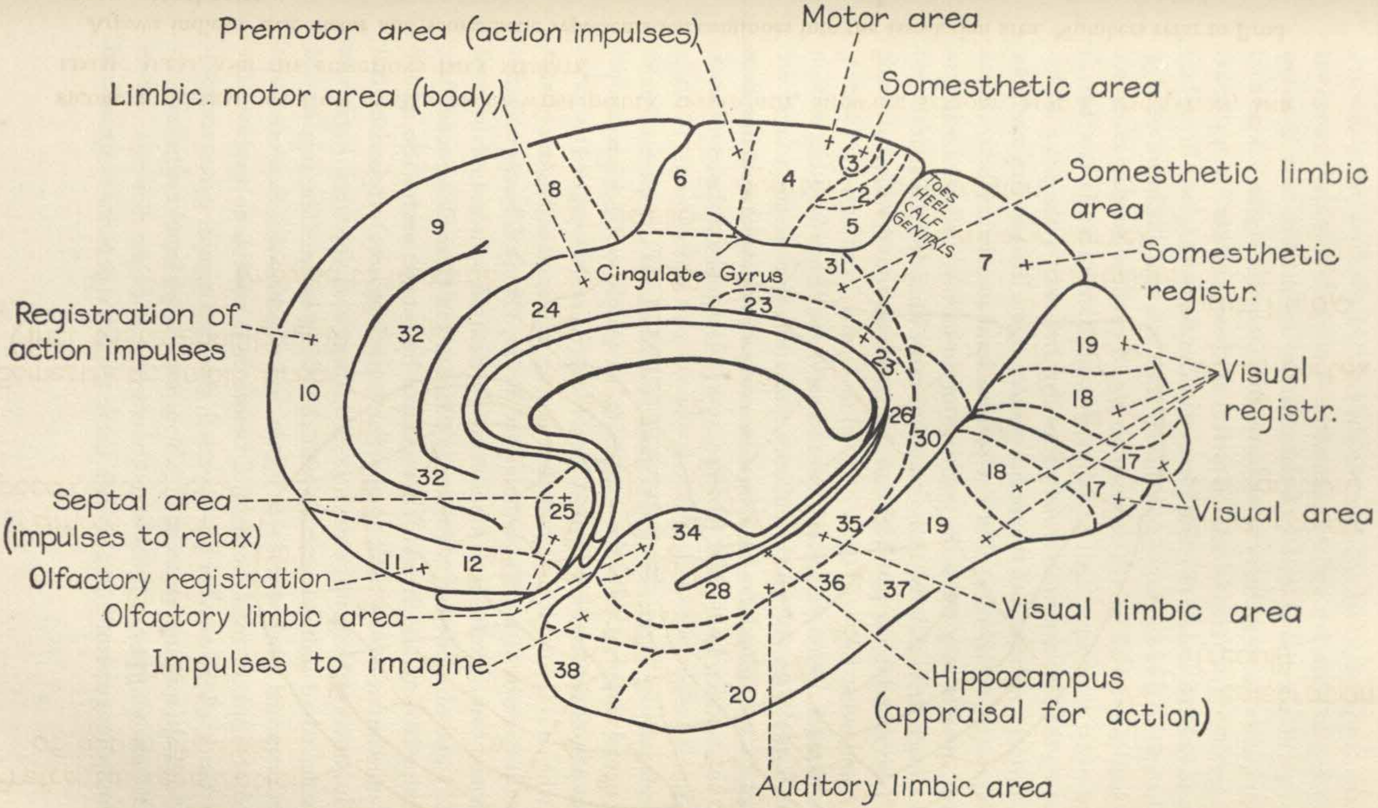

FIGURE 4. MEDIAL VIEW OF HUMAN BRAIN, SHOWING SENSORY, MOTOR, ASSOCIATION, AND LIMBIC AREAS, AND THE FUNCTIONS THEY MEDIATE

Numbers indicate Brodmann cortical areas.

superior temporal gyrus, dipping into the lateral fissure toward the insula. According to Pribram and MacLean, the superior and middle temporal gyrus excite the limbic cortex of the posterior insula. This would mean that impulses from auditory areas travel to this limbic region, mediating the appraisal of what is heard. On arrival of these impulses, things heard seem to be experienced as either pleasant or unpleasant.

The appraisal of intensity is possible as soon as a sound is heard. A sudden loud noise will be felt as disturbing and produce startle before we know what it means. This preliminary evaluation of sounds seems to occur via the *centrum medianum,* one of the thalamic intralaminar nuclei, which receives fibers from the medial geniculate nucleus carrying auditory impulses (cf. Peele, 1954). But one sudden loud sound may be a call to dinner, another may mean the approach of an enemy. To know whether to approach or withdraw, man or animal must know what has followed upon this sound in the past. He must remember the sound, what has made the sound, what followed upon it (food or enemy), and how he liked it. This requires not only appraisal but memory, and hence must depend on some cortical area. When the auditory and auditory association areas are destroyed, an animal can no longer be aroused by a voice call but does react to a sudden loud noise (Bremer, 1951; Fischgold and Lairy-Bounes, 1952).

Evaluation of sights. From Pribram and MacLean's work we know that neural impulses from the visual area are relayed to the hippocampal gyrus part of the limbic system. A visual impression is appraised as pleasant when it is favorable to smooth functioning. It is appraised as unpleasant when it disturbs visual functioning (see Vol. I, Ch. 4). The appraisal of high and low intensities as unpleasant (a flash of light or a faintly lit object) and of moderate intensities as pleasant can be made immediately anything is seen, and is probably mediated by connections to the medial thalamic limbic system. But to appraise something seen as good or bad for us, as something to be eaten or something that threatens harm, we must have had some experience with this thing before. The memory of what happened when it was seen before, and whether what happened was liked or disliked, must be recalled before it is possible to evaluate the object *as it affects me.* For this appraisal, the hippocampal gyrus seems to be essential (see our discussion of Klüver and Bucy's monkeys below).

Evaluation of tastes. It has been known for some time that taste nerves from the tongue run in the seventh nerve trunk (via *chorda tympani* and *lingula* nerve) and also in the ninth and tenth nerves, to the nucleus of the *tractus solitarius*. But the thalamic relay station and the cortical receiving zone have been established only recently. Taste fibers go to the thalamic arcuate nucleus which also receives sensory fibers from the face (Patton *et al.*, 1944). The cortical receiving zone is a small area on the lateral orbital surface of the parietal lobe, in the posterior operculum (Brodmann area 43). According to Ruch and associates (1952), the touch area for the tongue almost exactly coincides with the taste area, which explains the localization of taste in the somesthetic area.

Like every other sensation, taste also seems to be evaluated first as being adequate or excessive when impulses arrive in the nucleus *centrum medianum* in the thalamus, which receives fibers from the tenth nerve carrying taste impulses (cf. Peele, 1954). Different tastes apparently can be distinguished only when the cortical taste area is intact. They can be appraised as pleasant or unpleasant as long as this area is connected with the posterior insula.

Thus Penfield and Jasper (1954) found that a "bad taste" was felt when the border region between operculum and insula was stimulated electrically. According to these authors, both smell and taste sensations produced by electrical stimulation are invariably disagreeable. Electrical stimulation is more intense than sensory stimulation, which might account for this phenomenon. Also, electrical stimulation of the brain excites only the cell membranes, not the apical dendrites (Grundfest, 1957); hence, the sensations so produced will not have the range of sensations produced by sensory stimulation.

Bagshaw and Pribram (1953) found that monkeys refused quinine as long as the posterior insula was intact. Ablations limited to the anterior part of the insula and the anterior supratemporal plane did not affect their dislike of quinine. But after lesions extending into the posterior insula and operculum (see Bagshaw and Pribram's diagrams) the monkeys accepted a quinine solution 30 percent of the time. We believe that an ablation of the posterior insula alone (Brodmann's area 13 in man) would bring about the same inaccuracy in appraising something as unpalatable.

There is an interesting experimental report which seems to confirm our suggestion that things seen and heard are appraised, liked, or disliked via the hippocampal gyrus; things tasted, via the posterior insula; and things chewable, via the anterior insula (Brodmann area 14; see below, Evaluation of Eating). Klüver and Bucy (1939) found a remarkable change in the behavior of monkeys after ablation of the temporal lobes, including the hippocampus. These monkeys were apparently unable to recognize the things they encountered and mouthed everything repeatedly and indiscriminately, however often they investigated it. Their food habits changed completely. Instead of eating only fruits and nuts, as normal monkeys do, they ate anything edible, including all kinds of meat and fish. They were active all the time, running from one thing to another, smelling and mouthing everything in sight, whether it was animate or inanimate. They approached animals that had just attacked or injured them and even tried to mouth a burning match and the tongue of a hissing snake. For these animals, "certain properties of the objects, their being 'dangerous,' 'inedible,' or 'indifferent,' have suddenly become ineffective in determining visually guided reactions. The monkeys seem to be dominated by only one tendency, namely the tendency to contact every object as quickly as possible" (Klüver and Bucy, 1939, p. 988). These suddenly ineffective properties of the objects are not sense qualities; they require an evaluation of what is seen, guided by memory.

Such monkeys seem to see yet do not recognize objects. According to Klüver and Bucy, they are able to distinguish shape, distance, and brightness of objects, yet they do not seem to recognize what they see or to know what it is good for. They seem to hear, but they do not chatter at each other, nor do they join in the clamor of the monkey colony at feeding time. They have lost the limbic area for hearing and sight (the hippocampal gyrus) and have suffered damage to the neighboring insula. In addition, they have lost part of the hippocampus proper which is necessary for recall (see Ch. 3). As a result, they suffer from agnosia; they cannot recognize objects by sight or sound, and cannot appraise what they see or hear. The environment has become unintelligible. They are trying to orient themselves by smell alone; hence their "oral" behavior.

According to Klüver (1952), the monkeys eventually returned to their fruit and nut diet. Apparently, their taste discrimination was restored as soon as the remaining parts of the posterior insula and posterior hippocampus with hippocampal gyrus regained their normal function. In addition, the monkeys gradually stopped their "oral" behavior and reached for things with their hands, as do normal monkeys. As soon as their visual confusion cleared (as a result of the regained function of hippocampus and hippocampal gyrus remnants), they were able to judge the position and distance of objects and could reach for them.

Klüver and Bucy have also reported that monkeys without temporal lobes show greatly increased sexual activity, beginning about three to six weeks after operation. This is particularly noticeable in males who approach other monkeys, male or female, and indulge in sexual activity of a type never observed in normal animals. They show this symptom because they are unable to recognize and evaluate what they see. Moreover, the loss of the temporal lobe seems to prevent the activation of an image of mate and mating (see Chs. 3 and 6) and results in planless sexual activity rather than the normal mating pattern. That such hypersexed behavior appears three to six weeks after the operation can be explained by the cumulative sexual tension which builds up as a result of hormonal secretion and presses for release. Schreiner and Kling (1953) have shown in a comparable experiment that such unusual sexual activity disappears within a week after castration. Apparently, this behavior is caused by an accumulation of sex hormones.

A similar operation has been performed in human beings, in a vain attempt to make them less aggressive. When both temporal lobes were partially removed (including the uncus, amygdala, and anterior hippocampus, but sparing the middle and posterior hippocampus), the patients were unable to understand what was said to them and were unable to read, immediately after the operation. Some impairment apparently persisted for a considerable time (Obrador, 1947; Oldham, 1953). These patients showed a much slighter deficit than Klüver and Bucy's monkeys because only a small part of the temporal lobe was removed.

In another case, in which the anterior portion of both temporal lobes (including most of the uncus and hippocampus) was removed because of psychomotor attacks with grand mal epilepsy and dangerous aggressiveness, the operation resulted in a situation that almost duplicated what Klüver and Bucy had reported. The patient did not recognize anyone he saw and was completely indifferent to everyone and everything. At first, he was not very active, but after fifteen days he approached everybody passing by and picked up everything he saw to look at it. His attention could not be held by talking to him. In fact, he apparently understood very little of anything said to him. There was no aphasia, but his language was limited to a few elementary sentences. He could read but did not understand what he read. The only words he understood were "Mother," "Father," and "Verona" (his birthplace). He masturbated continuously and tried to have sexual contact with the men who talked to him. Before the operation he had shown no homosexual leanings. He ate voraciously, and was continually asking for food (and for cigarettes). He seemed to have no memories and no emotions, nor did he show any changes in facial expression, though his vegetative functions were unchanged. This state of affairs has persisted for two years to the time of reporting, and the patient has had to be committed to a mental hospital (Terzian and Dalle Ore, 1955). Neither this case nor the cases reported by Obrador and Oldham have come to autopsy. Hence, the extent of the operation cannot be checked, but there is little doubt that Terzian and Dalle Ore's was more extensive.

Evaluation of somesthetic impressions. A preliminary appraisal of organic sensations, and probably other somesthetic sensations as well, seems to be mediated by connections from the spinothalamic and trigeminothalamic tracts and the medial lemniscus to the nuclei of the midline and intralaminar nuclei in the thalamus. Thus various reflexes (deep and superficial) can be enhanced via the relays from these nuclei to the midbrain tegmentum and the action circuit (see Ch. 3).

The cortical area for sensations of touch, heat, cold, pressure, stretch, and various visceral sensations is in the postcentral gyrus which receives the bulk of the fibers from the spinothalamic and trigeminothalamic tracts and the medial lemniscus. Pribram and MacLean have shown that this area connects with the limbic cortex of the posterior cingulate gyrus, while McCulloch (1944) found that Brodmann's area 2 (in the postcentral gyrus) projects to area 31 (posterior cingulate gyrus). Therefore, localized somesthetic impressions can be appraised via the connections from postcentral gyrus to neighboring limbic cortex. Things touched, or localized organic sensations, can be felt as pleasant or unpleasant and can be liked or disliked when impulses arrive in the posterior cingulate gyrus.

According to MacLean (1954), electrical and chemical stimulation of the posterior cingulate gyrus has aroused increased pleasure reactions to petting in cats. On some occasions the animals showed sexual excitation also. This seems to confirm our suggestion that this region mediates the experience of pleasure from touch. Bard and Mountcastle (1947) reported a seemingly contrary observation, namely that cats showed increased pleasure after *removal* of the cingulate gyrus, subcallosal limbic area, and retrosplenial region. They purred, rubbed against the experimenter, and rolled on the floor when being petted. This effect seems to resemble the well-known hyperesthesia after destruction of the cortex (e.g., after hemidecortication) in man. It may be pleasurable or painful but is always diffuse and cannot be localized. It seems to depend on excitation from the somesthetic receptors relayed to the thalamic intralaminar or midline nuclei (cf. Head and Holmes, 1911).

Erickson's report (1945) of persistent "sexual" sensations in a woman patient, relieved after a tumor in the precuneus was removed, is indirect evidence for our interpretation also. Pribram and MacLean (1953) have found that the precuneus (area 7 in the somesthetic association cortex) is connected with the posterior cingulate gyrus, the parasplenial region and the posterior hippocampus. The tissue irritation in the precuneus of Erickson's patient seems to have tripped off nervous impulses to the neighboring limbic region mediating the experience of sexual pleasure. The precuneus is in the parietal association area at the level at which the genitals are represented in the postcentral cortex (see Fig. 4). The tissue irritation might have aroused a vivid somesthetic memory of sexual excitation which was felt as pleasurable and called a "sexual sensation."

Incidentally, Erickson's patient reported that this feeling was often so intense as to be almost painful—and Havelock Ellis, long before Freud, insisted that pleasure and pain are closely related in the sexual sphere. In Chapter 4 of the preceding volume, we have suggested that both pleasure and pain are feelings, reactions to optimal or excessive stimulation respectively. We have argued that pain cannot be a sensation as is usually assumed because it can accompany any somesthetic sensation yet varies independently. Now we can add that both pleasure and pain seem to be mediated by the medial thalamus (when diffuse) and by the limbic system (when differentiated and localized).

Pain accompanies tissue damage, but not all tissue damage is felt as painful. A growing tumor, tuberculosis, or hardening of the arteries may be completely painless. When the tissue damage becomes painful, the disease process seems to have attacked either the sensory pathways or their connections with the medial thalamus. When excitation in these relays becomes excessive, there is "thalamic pain." It may occur even after the somatosensory thalamic nuclei have been removed, for sensory pathways project to the reticular formation in the lower brain stem, and relays from there can reach the *centrum medianum* (one of the intralaminar thalamic nuclei) directly; the destruction of this nucleus abolishes pain when the destruction of the sensory thalamic nucleus has not been successful (cf. Bowsher, 1957). This is exactly what we would expect if the intralaminar thalamic nuclei mediated pleasure or pain as a reaction to somesthetic stimulation. In the intact organism, we think, tissue irritation in the sensory pathways will induce pain that is mediated both by intralaminar thalamic nuclei and the posterior cingulate gyrus. Destruction of the centrum medianum will abolish only the diffuse, persisting pain mediated by that nucleus, while destruction of the somatosensory thalamic nuclei will abolish the sharp, localized pain but leave the diffuse pain untouched. As Bowsher has reported, both nuclei have to be destroyed if the pain from irritation of the sensory pathways is to be prevented.

Since pain seems to be a reaction to excessive somesthetic stimulation, the congenital insensitivity to pain found in isolated cases might be the result of a paucity of functioning cells either in the estimative or in the somatosensory system. We would suspect a reduction of functioning sensory cells when somesthetic sensitivity is reduced (e.g., in a case reported by Cohen *et al.*, 1955); and a defect in the estimative system when touch and other somatic sensations are unimpaired (e.g., in a case mentioned by Lüthy, 1957). Since free nerve endings were found in the patients mentioned by Lüthy wherever the skin could be explored, it is unlikely that free nerve endings are the receptors for a simple sensation of pain. Barber (1959) also has come to the conclusion that pain is not a simple sensation.

Evaluation of smell. The olfactory system is the only sensory system which avoids the thalamus altogether. But it contains structures that have substantially the same functions as those in the brain stem and thalamus. From receptors in the upper part of the nasal cavity, olfactory nerves go to the olfactory bulb, connect with the anterior olfactory nuclei (corresponding to brain stem nuclei) and the olfactory tubercle. This may be the receiving area for smell and may be a rudimentary type of cortex (Crosby and Humphrey, 1941; Clark and Meyer, 1947). Since smell is diffuse, and unlocalized, the olfactory tubercle may correspond to the thalamic sensory nuclei. It sends relays via the medial olfactory striae to the medial orbital cortex (which may be the olfactory association area) and to the subcallosal gyrus; and via the lateral olfactory striae, to the prepyriform region. The subcallosal gyrus and the prepyriform area are part of the limbic region. The olfactory tubercle also connects with the anterior part of the hippocampal rudiment via the medial olfactory stria (Clark and Meyer, 1947; Meyer and Allison, 1949; Allison, 1954).

Any odor seems to be sensed via the olfactory bulb and tubercle, and appraised via the anterior olfactory nucleus and the olfactory limbic cortex. If the odor is very strong or pungent, it is felt as unpleasant and may even become painful. Pungent odors (e.g., ammonia) affect the trigeminal nerve (Elsberg *et al.,* 1935). The discomfort or pain aroused by it seems to be a reaction to the somatic rather than to the olfactory sensation. However, an odor may be disliked quite apart from its somatic effects. It may arouse an immediate feeling of unpleasantness or it may be disliked because of some association with a former unpleasant experience.

Allen (1940, 1941) found that ablation of the hippocampus did not interfere with a conditioned olfactory response and concluded that the hippocampus is not an olfactory structure. Prefrontal lobectomy, however, prevented dogs from learning a conditioned olfactory response (withdrawing the foreleg upon noticing the smell of cloves which announced an electric shock). It also prevented them from learning to discriminate between the smell of cloves, which announced a shock and required withdrawal of the foreleg, and the smell of asafoetida which did not announce such a shock. This means that the dogs could not remember that the smell of cloves is different from the smell of asafoetida. But they could and did select a packet of meat from a collection of packets containing inedible things. Since they could not distinguish between smells, they could not have recognized the meat by smell. But they apparently could appraise this smell as good, an appraisal that seems to be mediated by the anterior olfactory nucleus.

Pechtel and associates (1955) reported a similar result in cats. Destruction of the dorsomedial thalamic nuclei (which project to the prefrontal cortex) abolished a conditioned olfactory discrimination. In months of retraining, these cats did not learn to distinguish between different smells, but they could follow the smell of meat or fish. However, when meat, fish, or milk were mixed with oil of wintergreen or mephenesin powder (both smells intensely disliked by cats), they would no longer touch the contaminated food though they had readily taken it before the operation. The animals, like Allen's dogs, could appraise a smell as pleasant or unpleasant, but they no longer expected that an unpleasant smell might be attached to palatable food.

Evaluation of action: Body movement. According to Pribram and MacLean, the superior frontal neocortex (motor and premotor areas) is connected with the anterior cingulate gyrus (see Fig. 2). Actions or single movements seem to be appraised when relays from the motor areas arrive in the anterior cingulate gyrus and so can be experienced as suitable or unsuitable. There must be some way in which an animal can evaluate its actions: a cat, finding itself locked up in a cage, will climb up the bars, try to dig around them, or to squeeze through. Whenever one method proves futile, the cat tries something else. To abandon one action in favor of another, man or animal must recognize it as unsuitable or unsuccessful. This recognition is not a sensation like red or blue or sweet or rough. It is a *sense judgment* evaluating how this thing or action *affects him here and now*. Such an appraisal is not reflective, it is a mere awareness that this is good to do; this experience is quite comparable to the experience of finding something good to smell, to hear, or to touch, and liking it. Apparently, this experience is mediated by the limbic region nearest the motor region, namely the anterior cingulate gyrus. The experimental evidence showing the effect of the destruction of this area is quite convincing. Ward (1948), in a much-quoted passage, describes the far-reaching behavior changes thus:

> The monkey loses its preoperative shyness and is less fearful of man. It appears more inquisitive than the normal monkey of the same age. In a large cage with other monkeys of the same size, such an animal shows no grooming behavior or acts of affection towards its companions. In fact, it treats them as it treats inanimate objects and will walk on them, bump into them if they happen to be in the way and will even sit on them. It will openly eat food in the hand of a companion without being prepared to do battle and appears surprised when it is rebuffed. Such an animal never shows actual hostility to its fellows. It neither fights nor tries to escape when removed from a cage. It acts under all circumstances as though it had lost its "social conscience." (1948, p. 440)

Such monkeys seem to differ considerably from the monkeys in Klüver and Bucy's experiment. After hippocampal destruction, these monkeys could no longer appraise what they saw or heard. They could no longer recognize their environment except by touch and smell. But Ward's monkeys seemed to have no difficulty in recognizing everything around them. Their difficulty was rather that they were unable to gauge the effect of their actions—they always seemed to do what their impulse urged them to do without realizing or caring what their action might lead to. Thus they took food wherever they found it, even out of the hand of another monkey; they sat wherever they wanted to, even on one of their fellows, apparently oblivious of the consequences. They did not fight when disturbed because they could not gauge the effect of such a move either. This behavior is exactly what we would expect if the anterior cingulate gyrus served the appraisal of action or action impulses. Similarly, if an animal cannot assess the effect of grooming, he will make no attempt to groom either himself or his fellows. Ward's monkeys apparently were unable to recognize when anyone interfered with their actions or to gauge the possibility of escape, hence their curious lack of any move toward fight or flight.

Klüver and Bucy's monkeys, on the other hand, were well able to appraise any interference with their actions or intentions. They became angry, for instance, when the floor was swept or a wagon was rolled by near their cage, but their anger disappeared as soon as they were allowed to investigate broom or wagon. Apparently, they became angry whenever they could not investigate something they did not recognize by sight, although they were well able to recognize their frustration. But they did not show anger when threatened with a stick because they could not identify what they saw. Interference with motion, according to Klüver and Bucy the chief cause of anger in their experimental animals, can evidently be recognized and appraised as long as the anterior cingulate gyrus is intact, while the appraisal of things seen or heard requires a functioning hippocampal gyrus.

Pribram and Fulton (1954) have recently published an "experimental critique" of the deficit following destruction of the cingulate gyrus. They trained monkeys on a visual discrimination problem in which a cup bearing a cross was baited behind a screen. The monkeys had to choose between this cup and an empty cup bearing a square. After removal of the cingulate gyrus or the medial frontal cortex on both sides this habit was unimpaired, and Pribram and Fulton suggest that there is no deficit after such operations. Actually, their experiment only shows that a *visual discrimination habit* is undisturbed by anterior cingulate ablation; but the changes found by Smith and Ward were not the result of a defect in visual discrimination. To find the defect, a problem would have to be chosen in which the monkeys would have to appraise the effect of some action of theirs. If our suggestion is correct, they would not succeed in a problem in which they would have to cooperate with another monkey, nor would they be able to avoid a tipping plank on a runway.

Our suggestion that the anterior cingulate gyrus serves the appraisal of action seems to be supported by stimulation experiments also. Ward (1952) stimulated the anterior cingulate area in unanesthetized cats and found that the animals stopped walking within two to three seconds (with near-threshold current stimulation) and more quickly if higher intensities were used. Sometimes they simply stood still, at other times they turned their head to the opposite side and slowly lowered their tail. With stronger stimulation, they might crouch, but if they saw a mouse they pounced in spite of such stimulation. These cats remained alert throughout, turning their heads toward unusual sounds and arching their backs when stroked. However, when stimulation of the motor cortex produced movement, additional stimulation of the anterior cingulate gyrus facilitated such movement. The spontaneous electrical activity of the motor cortex was neither suppressed nor changed by stimulation of the anterior cingulate gyrus.

These findings cannot be explained by the usual notion of the cingulate gyrus as a "suppressor area" but become intelligible if we assume that this area serves the experience of a given movement as suitable or unsuitable. The cat stops moving because electrical stimulation of the anterior cingulate gyrus excites cells receiving relays from the premotor and motor areas which make movement feel inappropriate or unsuitable. When the motor cortex is stimulated as well, the induced movement will find no resistance because the animal's own movement has stopped, and the electrically produced movement will appear to be facilitated. Excitation of the anterior cingulate gyrus, relayed from the motor area or directly from the thalamic limbic system, apparently can either inhibit or facilitate action, depending on the appraisal of such action as appropriate or inappropriate. If appropriate, the action will be felt as pleasant and will continue. If inappropriate, it will be felt as unpleasant and there will be no impulse to continued action; rather, all ongoing action impulses will be inhibited. Since electrical stimulation is different from stimulation via afferent relays (see above), such stimulation will generally induce the appraisal that movement is unsuitable, and result in arrest of action. But when a strong action impulse is aroused (when a cat sees a mouse running by) action will be immediate and "break through" the inhibition. Every action impulse has to be felt as good, or it would not even become an impulse to action. Such preliminary appraisal, we think, is mediated by the medial thalamus which, according to Sloan and Jasper (1950), is connected with the anterior cingulate gyrus.

Evaluation of action: Eating and speaking. According to Pribram and MacLean, the anterior frontal operculum and the strip of premotor cortex just above are connected with the anterior insula. We shall see later (Ch. 3) that the premotor cortex mediates impulses to move and relays them to the motor cortex. In this premotor strip, the legs, trunk, and arms are represented on the dorsolateral surface (see Fig. 3), while mouth, larynx, and pharynx are represented on the lateral base of this strip and in the operculum.

According to our scheme, movements of mouth, tongue, larynx, and pharynx in eating or talking would be experienced as suitable or unsuitable when impulses arrived in the anterior insula. Depending on this appraisal, eating or speaking would either be continued or halted. Electrical stimulation of this region, particularly the anterior insula, has actually produced chewing, licking, and swallowing (Fulton, 1951a). If an animal were so stimulated while eating, we would expect that excitation of this limbic area would now be felt as excessive and eating would stop.

When a man or an animal wants to eat, he must estimate whether a likely looking object is edible. Once he has started to chew, he must gauge the effect of his efforts. On finding a piece of metal or a stone in his food, for instance, he will realize that this cannot be cracked or chewed and will stop. Either to start or to stop eating, man or animal must be able to appraise the effect of his effort. A report by MacLean (1952) illustrates what happens when such appraisal is no longer possible. After bilateral ablation of the anterior temporal lobes the patients developed a curious tendency to eat things usually considered unpalatable. One patient, for instance, was eating potato chips from a bag, and when he had finished the chips, he ate the bag, too. Others were eating toilet paper, orange peels, and similar things, actions that strikingly resemble the indiscriminate feeding habits of Klüver and Bucy's monkeys after ablation of the temporal lobes. In all these cases, the insula was damaged and, at least for a time, prevented from functioning normally. As a result, their own activity of eating could not be evaluated adequately. Significantly, there was one monkey in Klüver and Bucy's experiment who did not eventually return to the usual fruit diet as did the other monkeys. This animal had been deprived of both frontal and both temporal lobes, and so had lost the anterior insula on both sides. It could no longer recognize food by smell, nor could it appraise what is good to chew.

In this brief survey we have tried to show that every sense impression and every action impulse must be evaluated as good or bad, harmful or beneficial. The preliminary appraisal of sensations and action impulses seems to be mediated by relays to the medial thalamus, while the appraisal and identification of objects, the appraisal of an impulse to directed action and of the action itself, seems to depend on relays to the limbic cortical areas. The experience of liking or disliking what is so appraised seems to be mediated by the medial thalamic areas connected with the sensory pathways over which a given impression is received, and by the cortical limbic areas nearest the sensory areas that mediate object perception.

An intuitive appraisal of something touched or tasted is the immediate reaction to the effect of this thing on us, even before it can be identified. So we quickly pull away if we have touched something hot, and we spit out anything that we cannot chew or that has an unpleasant taste. Similarly, the intuitive appraisal of a movement is the simple reaction to the effects of moving. So a man will jump aside when stepping on yielding ground or will try to recover his footing when slipping. But to appraise the effect of something seen, heard, or smelled, or to appraise the eventual consequences of an action, we must compare the present situation with a similar situation in the past. The so-called "distance receptors" allow the appraisal of things perceived only after we compare what we see, hear, or smell with the remembered consequences of similar sights, sounds, or smells.

Appraisal for action. When a child sees a man approaching him, he will immediately remember what such an approach has meant in the past. If the man is a physician who has poked and prodded the child before, and perhaps has given him a painful injection, the child will remember these past experiences on seeing him, will expect to be hurt again, hate the thought of it, and want to run away. To experience fear on seeing this man, the child must remember that this man has hurt him in the past and judge that he will do so again. Hence fear implies a complex combination of sense impressions and memories.

Even when memory is not yet available, as for the newborn infant, there must be a sequence of sense impressions, appraisal, and emotion (i.e., liking or disliking something together with an impulse to approach or avoid it) before there can be action. Even the neonate has some notion of what is good and hence wanted, and what is bad and hence to be avoided. The infant experiences hunger (i.e., an impulse to action) and seeks with his mouth until he encounters a nipple; then he sucks lustily and swallows the milk. If it is not milk but vinegar he spits it out and turns away. These are not mechanical reflexes.* They are actions that imply wanting, appraising, liking, and disliking. Even to stop nursing implies a recognition that no more is wanted, and an impulse to stop.

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* If they are considered mechanical connections between sensory and motor nerves, it is difficult to explain why such reflexes come to be invested with

There is a definite sequence of psychological experience and action in such behavior that requires a circuit mediating these events in this particular order. There is always liking or disliking as a reaction to sensory impressions, which leads to an impulse to a definite action. There must be a structure that will collect impulses from sensory areas and relay them to the motor cortex where the urge to action can be experienced. A transcortical relay will not do because various sense impressions must first be combined, then compared with relevant memories, and finally assessed for possible action. Strictly intracortical connections run between neighboring cortical regions (short association fibers) or between regions farther apart (long association fibers), but there is no known intracortical circuit that would make it possible to collect neural impulses from every sensory area, combine them, reactivate impressions which have been registered before, and finally mediate appraisal, liking or disliking, and the emotional impulse to action, in that order.

There is a system that could provide such a circuit via subcortical structures, the *hippocampal system*. This system spans the inner border of the hemisphere from the septal area in the frontal lobe around the *corpus callosum* to the hippocampus proper, which extends throughout the temporal lobe (Figs. 4, 5). Impulses from each sensory area, relayed to the adjoining limbic region, can be sent to the hippocampus where they can be collected and relayed via the main efferent pathway, the fornix, to the hypothalamus, midbrain, and eventually, as we shall see, to the frontal lobe (see Ch. 3). But the impulses collected in the hippocampus can also be relayed via the midbrain to the association cortex and via the medial thalamic system to the whole cortex (see Chs. 3 and 5).

The hippocampal circuit seems to be active whether an object can be appraised immediately or must first be identified by recalling earlier experiences with it. The hungry infant, for instance, does not need memory to feel hunger and want to suck. But he must be able to judge that what he touches and seizes with his lips is good to suck; and also, that the milk he has in his mouth is good to swallow. He will not suck something sharp

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feeling in adulthood. It is even more difficult to sketch a neural mechanism that would bring it about at such a late stage. We hope to show in a later chapter that even spinal reflexes include a primitive appraisal of the effect of sensory stimulation on the organism (see Ch. 5, The Peripheral Estimative System).

and cold nor will he swallow something sour or bitter. Accordingly, he must be capable of an appraisal which is not only immediate and intuitive but *innate*—in the sense that the human infant (like every other infant mammal) is so constituted that something sweet will be liked and wanted, and will arouse an impulse to swallow; that something of the right consistency touched with his lips will feel good-for-sucking, and that hunger will arouse an impulse to seek with his mouth until he finds a nipple. All instinctive actions imply such an intuitive innate assessment of the things necessary for the well-being of the individual (or the species); they are ordered to his use.* The same innate appraisal will occur when he meets things that harm him, and will arouse an impulse to avoid them or fight them.

But not every appraisal is innate, nor is every action instinctive. Which of all the things in his environment are good for a man or an animal, which bad, he must learn by experience. Eventually, he is able to avoid what can harm him before he is actually hurt and can pursue what he wants before it escapes him. Both fear and desire depend on memory. Having no memory, the neonate is without fear and shows no joy on seeing either mother or bottle. As he begins to experience what people and things in his environment do to him and what he can do in turn to get what he wants and to avoid what he dislikes, he gradually learns to identify and appraise his world correctly and to act accordingly. Once he has felt pain inflicted by someone or something, he will remember what led to it and will begin to be afraid of that person or thing. Fear always depends on the memory of past harm and the expectation that similar situations will bring similar hurt. Even so-called "instinctive" fears in animals, or the "fear of the unknown," depend on the felt contrast with what is known to be friendly (see Vol. I, Ch. 12).

MEMORY TRACES AND THEIR LOCATION

Several authors have suggested that the hippocampus may be a memory mechanism, for bilateral degeneration of this structure is known to result in severe memory disturbances (Ule, 1951; Kaada *et al.,* 1953). Such defects also occur in Korsakoff's psychosis, which is accompanied by isolated destruction of the mammillary bodies, the end station of relays from the hippocampus via the postcommissural fornix. Milner and Penfield (1955) and Scoville and Milner (1957) have reported severe and lasting loss of "recent memory" after bilateral removal of the hippocampus and hippocampal gyrus, or of the lateral temporal cortex, or of both together. Unilateral ablation, or bilateral removal of the anterior four centimeters of the temporal lobe, may cause some temporary memory loss, but no lasting defect. On the other hand, Dott (1938) and Garcia Bengochea *et al.* (1954) have reported that cutting the fornix on both sides has produced no untoward effects, neither memory loss nor any other deficit.

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* "Imprinting," the characteristic following response to the first moving object a chick or duckling encounters (see Tinbergen, 1951; Lorenz, 1952), also presupposes such an innate appraisal, an estimate that "this is good to follow." This appraisal leads to action as soon as chick or duckling is able to move at the necessary speed.

To resolve these contradictions, let us try to discover the role of the temporal lobe cortex, as well as that of the hippocampus and fornix, in the registration and recall of memories. In recent years, many investigators have found that lesions of different cortical areas have different effects on learning; and learning, let us note, always implies memory. Among psychologists, it has usually been held that memory (and therefore learning) is a unitary function mediated by the brain as a whole ("mass action"; cf. Lashley, 1929, 1950). But the evidence that has accumulated in recent years seems to suggest, rather, that the sense impressions in each modality are registered separately. Hence memory is not a unitary function. It includes *visual* memory, mediated by the visual association cortex, *somesthetic* memory mediated by the somesthetic association area, and *auditory, olfactory, gustatory,* and *motor* memory mediated by their respective association areas. Nielsen (1956), among other neurologists, has held this view for years.

That this is the only possible inference is suggested by many recent findings. In a series of ingenious animal experiments, the Yale group has shown that a learned *visual discrimination habit* is lost after ablation of the inferior edge of the temporal lobe and the preoccipital cortex (Pribram and Mishkin, 1955; Pribram and Barry, 1956); a learned *auditory discrimination habit,* after removal of the posterior temporal cortex (Weiskrantz and Mishkin, 1958); a learned *somesthetic discrimination habit* (both touch and weight discrimination), after loss of the parieto-occipital association area (Wilson, 1957). A learned *alternation habit* was lost after destruction of the lateral frontal cortex (Mishkin and Pribram, 1955), and conditioned *taste discrimination,* after destruction of the somesthetic association area for the tongue (Bagshaw and Pribram, 1953). From Allen's (1940) findings and the known distribution of olfactory fibers we may conclude that the orbital cortex is necessary for conditioned *olfactory discrimination.*

It is true that the above authors do not themselves suggest that their ablation experiments resulted in *memory* loss. Instead, they speculate that "attention" may be lost, or that the relevant cues are no longer "distinctive." But Chow and Orbach (1957) have shown that this is not a deficit in attention. And the lost "distinctiveness" means in the last analysis that the animal can no longer *remember* that this particular thing has indicated food in the past. Perhaps the objective terminology so popular among experimental psychologists has prevented them from recognizing that a discrimination habit cannot be established unless the animal remembers that this thing he sees or hears or touches has indicated food in the past, or that this particular movement has led to food before. Similar injuries have led to similar memory defects in man.

Visual memory. There is considerable evidence that visual impressions are registered in an extensive area of the occipital, parietal, and temporal association cortex in man. The visual *recognition* of objects seems to be mediated by Brodmann area 18 (see Figs. 3, 4). According to Nielsen:

> If the cortex of area 18 is destroyed . . . the patient has complete visual agnosia for objects. He sees well enough to pick up a pin from the floor but he does not recognize anything until he feels, hears, tastes or smells it. He is lost in his own house but can feed himself if placed at the table and told that what is placed before him is good to eat. . . . But the patient still can read and calculate. (Nielsen, 1943, p. 235)

Visual imagination, however, and the *recall* of visual images, seems to require an intact area 19. After destruction of this area, the patient can still recognize wife or husband, his house or pets, his town or neighborhood, but he can no longer visualize them when he is away from them. When asked, he cannot tell the color of a canary or of the hair of husband or wife, and cannot describe his house or street or town unless he actually sees them. He is no longer able to draw on his visual memory or to imagine anything visually.

Wilson and Mishkin (1959) found poor "discrimination" of size and pattern after lateral occipital lesions in monkeys, though these animals showed good visual learning. Monkeys with inferotemporal damage showed poor visual learning but fairly good discrimination of size and pattern. Visual learning implied *recalling* that one object had announced food, another no food, while visual discrimination meant *recognizing* a disk or *recognizing* which string had a peanut attached to it. Apparently, there is a distinction between the areas necessary for recall and those needed for recognition, in animals as well as in man. The lateral occipital region seems to be homologous to area 18 in man, serving recognition, while the inferotemporal region seems to be homologous to area 19 in man, serving recall or visualization.

In addition to the greater size of the visual association area in man, corresponding to the greater range of memories (words and symbols in addition to objects), there seems to be unilateral dominance in man which has not been found in animals (Nielsen, 1943). For instance, McFie and Piercy (1950) reported their findings on eight patients who suffered from gross difficulties in recognizing objects and spatial relations, and came to the conclusion that such lack of recognition (visual agnosia) is the result of damage to the minor hemisphere.

Auditory memory. According to Nielsen (1946), the posterior half of the superior temporal gyrus (Brodmann area 22) and part of the middle temporal gyrus (area 21) are needed for the comprehension of spoken language. The anterior part of the superior temporal gyrus seems to serve the recognition of music.

The visual and auditory agnosia of Klüver and Bucy's monkeys after temporal lobectomy shows that temporal association areas are needed for visual and auditory recognition. According to Klüver (1952), these monkeys later relearned visual discrimination. This is possible because visual impressions seem to be registered in the occipital as well as in the inferotemporal association cortex, and can be appraised via the remaining posterior hippocampal gyrus.

Clearly, agnosia is far more complex in man because he must be able to recognize written, printed, and spoken words as well as actual objects. Besides the simple inability to recognize written and printed words (alexia) and the inability to recognize spoken language (sensory aphasia, or better: auditory aphasia), there may also appear an inability to understand what words mean (semantic aphasia or "mind blindness"). We would suggest that this occurs when there is a lesion of the hippocampus that prevents the patient from recalling to what object a word refers; or when there is a lesion in the insula, so that he can no longer evaluate these meanings. According to Grinker and Bucy (1949) and Nielsen (1946), a deep lesion in the insula, or in the neighborhood of the angular gyrus, results in this type of aphasia. Without the insula, sounds can no longer be appraised. And a deep lesion, either in the insula or near the angular gyrus, damages the hippocampus at its junction with the fornix; hence the patient can neither identify what he sees or hears, nor can he formulate words or sentences.

Somesthetic memory. Tactile agnosia, that is, the inability to recognize things by touch, has been reported after lesions of the postcentral gyrus, the superior parietal lobule, or the supramarginal gyrus. After large lesions of the inferior parietal lobule, including areas 39 and 40, the patient may be completely unaware of his own limbs on the side opposite the lesion. He may not recognize hand, arm, or leg as his, or he may not know that he is paralyzed. On being asked to move his leg, he may go through the psychological process of moving it and state that he has moved (Nielsen, 1946). When the somatosensory cortex is removed, but the association cortex in the parietal lobe is intact, there is no such delusion. Apparently, the lesion that has resulted in agnosia has destroyed the area in which somatic impressions are registered. Touch, pressure, or movement can be felt but neither recognized nor recalled. If a man cannot recognize sensations from his limb, he will not realize that the limb is his. Vision can bring a reflective judgment that this arm is connected to his body and so must be his, but it cannot replace the recognition and recall of somatic sensations.

Olfactory memory. From reports mentioned before, we have concluded that olfactory impressions must be registered within the projection area of the dorsomedial thalamic nucleus, probably in the orbital cortex. In human beings, a similar loss of olfactory memory has been reported after prefrontal surgery. After venous ligation, transorbital lobotomy, or destruction of the dorsomedial thalamic nuclei, patients could no longer recognize various odors, though the olfactory threshold was unaffected. Some patients reported the same odors in all trials, even though three different substances were used (coffee, phenyl ethyl alcohol, and oil of peppermint). Other patients named colors instead of odors, and still others reported a stronger odor as a different odor (Wenzel, 1952). We have been able to verify this memory loss after standard lobotomy. Psychotic patients showed a complete loss of olfactory memory six years after the operation, while a control group of psychotic nonlobotomized patients recognized the odors as easily as did normal people (Caldwell, 1958).

Taste memory. Bagshaw and Pribram (1953) found that lesions limited to the parietal operculum did not affect monkeys' dislike of quinine. But it seems likely that such a lesion would interfere with taste memory even though it leaves the appraisal of tastes unimpaired. An experiment in which a sweet or salty taste is used to announce an electric shock should decide this point. As long as taste memory is intact, an animal should be able to distinguish salt from sugar and should also be able to distinguish different degrees of saltiness or sweetness. We believe that such discrimination would be impossible after ablation of the parietal operculum; but the appraisal of what is palatable or unpalatable (e.g., quinine) seems to be impaired only after ablation of the insula.

Motor memory. We do not only remember what we have seen or heard or felt, we also remember what we have *done* in a particular situation. Indeed, we must be able to remember earlier actions and to imagine what is to be done here and now before we can decide on action.

Mishkin and Pribram (1955) have reported that destruction of the lateral frontal cortex in monkeys results in the loss of an alternation habit. These animals had learned to go to the right cup if last time they had found food in the left cup, and vice versa. After the operation, they were unable to respond correctly, apparently because they could no longer remember to which cup they had gone in the previous trial. Hence, it would seem that the memory of movements and actions is mediated by the dorsolateral frontal association areas.

This interpretation was confirmed by a later experiment (Pribram, 1956) in which the animals responded correctly when the experimental conditions were changed in such a way that they now had to remember what they had *seen* before: if a tobacco tin was put on top of the baited cup and an ash tray on top of the empty cup, the animals chose the correct cup, indicated by the tobacco tin, whether it was right or left, above or below the empty cup, identified by the ash tray. After prefrontal ablation, monkeys can apparently alternate between two cups when they have to recall what they have *seen* the last time, but cannot do so whenever they have to remember what they have *done*.

However, when six cups were arranged in a circle and the tobacco tin was placed in random order over one of the cups to indicate that it was baited, normal animals and animals with inferior temporal ablations chose the correct cup in 75 percent of trials (after 250 trials), while animals with prefrontal lesions were still choosing by chance. To explain this defect, we have to remember that this experiment requires that the correct cup be chosen out of six possible cups, all arranged in a circle. The monkey must remember the correct object, look around, find it, and then act. This looking around seems to require a realization of *what is to be done,* that is, motor imagination. After the loss of motor memories, motor imagination also is impossible, hence the deficit.

Another study shows a similar defect in motor imagination after destruction of the dorsomedial nucleus on both sides. After the operation, cats completely lost a complicated habit of manipulating switches to obtain a food reward. In several months of retraining, they could not relearn the habit. They never even pressed the switch (step 2 of a seven-step performance) unless it was baited, though all intact animals pressed an unbaited switch after a few trials (Pechtel *et al.,* 1955). It is easy to see that the cats could not *imagine what to do* to obtain food; when the food was on the switch, they pressed it to reach the food—which requires no motor imagination.

In human beings also, injuries of the prefrontal areas result in similar defects. To make a coordinated movement, for instance, in writing a word, we must first have a picture of the word as it is to be written and next must be able to execute this movement smoothly and quickly. Practice is necessary to achieve such motor coordination; and the pattern of the movement, we believe, is preserved in the frontal association cortex. Once learned, the impulse to move is reinforced by neural impulses from that pattern. When someone who is right-handed tries to write with his left hand, he will find it impossible to form the letters correctly, though he knows how they should look. Since the left motor area is dominant in right-handers, the right motor association area does not have the necessary motor engrams. From the evidence it would seem that the motor engrams for the various body regions are registered in the same cortical plane as the motor representation in the cortical motor area (see Fig. 3). Motor engrams for knee and leg would be represented in the superior frontal gyri, while face and throat seem to be represented in the inferior frontal gyri, both in motor and association cortex.

The so-called "writing center" seems to be in the posterior part of areas 8 and 9, namely in the middle frontal gyrus, at the level of the motor representation of the hand and arm. When this area is damaged, the patient is unable to write spontaneously, though he is able to copy what is put in front of him (Nielsen, 1941). We would suppose that such a patient can no longer imagine how to move his hand to form the letters, and so cannot write spontaneously; but he can copy, for motor facilitation (motor recognition) is intact.

Broca was the first to suggest that there is a "speech" area in the inferior frontal gyrus (area 44). When it is destroyed in the dominant hemisphere, the patient is unable to speak correctly though the speech muscles are not paralyzed and can be moved at will. When he is asked to repeat a word, he can often do so (Nielsen, 1941). This seems to mean that he can no longer imagine what is to be said, how to use his vocal organs spontaneously, but can do so as soon as he knows what to say.

Area 45, the triangular part of the inferior frontal gyrus, seems to be active in registering movements made when singing or when playing wind instruments. After a lesion in this area, the patient is no longer able to sing; after a larger lesion, extending to the more dorsal region, he cannot play a musical instrument either (Nielsen, 1941).

In all these cases, a defect is apparent only when the dominant side of the brain is damaged. Even so, the patient can be retrained, that is, he can acquire new motor memories that are now registered on the nondominant side. Nielsen reports that one patient, whose speech area was damaged on the dominant side, reacquired speech after a short time. But some years later, a hemorrhage destroyed the same area on the other side. This time the patient remained mute for the rest of his life.

In all these cases, it is motor imagination as well as motor memory that is lost. The patient cannot imagine what to do spontaneously but can imitate someone else, probably because motor facilitation depends on intracortical relays from association areas to motor cortex and so is never entirely lost. For this reason, prefrontal lobotomy does not result in an obvious defect, though tests that depend on motor imagination, as, for instance, the Porteus maze test, do show a deficit. Few of our psychological tests explore motor imagination in a way that would show such impairment. Nielsen mentions that a lesion of the frontal lobes may produce disorientation in space. He says: "The patient is lost in his environment, but, on questioning, it is found that he can give an accurate revisualization of directions and distances; he merely does not know how to use the information" (1941, p. 282). He cannot imagine *what to do* to reach a particular place which he can visualize perfectly. Brain damage in the frontal lobe apparently interferes with motor imagination.

There is considerable evidence that neighboring parts of each sensory association area can mediate the registration of sense impressions when the main region is damaged. Thus Pribram and Mishkin (1955) have found that lost visual, auditory, or somesthetic discrimination habits can be reacquired by brain-injured monkeys, provided they are given a great deal of additional training. Klüver and Bucy's monkeys also could be retrained in visual discrimination after removal of the greater part of the temporal lobes.

From this short survey we may conclude that the registration of sensory impressions and movements occurs in the cortical areas close to the primary sensory and motor areas, usually called the "association cortex." These memory traces can either be activated as a structural whole (a memory) or recombined into various new structures (images). They seem to be activated as memories when a new sense impression calls for interpretation. They must be combined with all available sense impressions if man or animal is to connect what he sees with what he remembers of similar situations in the past and their effect on him—if, that is, the situation he encounters is to become meaningful to him.

3. FUNCTIONAL SEQUENCE: II. THE IMPULSE TO ACTION

A few impressions can be appraised for action immediately. These are the result of excessive somesthetic or taste stimuli: a blow, a cut, a bitter taste. But in most cases we must recognize the object or situation and compare it with similar situations in the past and their effect on us before we can take appropriate action. Such recognition and recall lead to the identification of object or situation as something that can harm or benefit us, and lend meaning to our experience.

THE ACTIVATION OF MEMORY TRACES

Both recognition and recall imply an activation of memory traces. What has been registered must be revived and reexperienced. This can be done spontaneously when an experience triggers off a relevant memory. A memory can also be recalled deliberately, at least by the human being. There is, however, a difference between recall and recognition, between bringing to mind something experienced before and having a feeling of familiarity when we see or hear something we have seen or heard before.

Recognition. Corresponding to this difference in experience there is a difference in the cortical areas required for recognition and recall. The areas for recognition (e.g., area 18 in the visual cortex, area 42 in the auditory region) seem to be secondary sensory areas * bordering on the primary areas. To recognize something as, for instance, having seen it before seems to imply that the secondary sensory area has been activated more than once in the same way. The second time this particular neural pattern arrives in the primary visual area (Brodmann area 17), it will mediate, as before, the experience of "seeing this thing"; but the second time the pattern arrives in the secondary area (area 18), it seems to give rise to the experience of "having seen this before."

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* These secondary sensory areas should not be confused with Woolsey and Fairman's (1946) and Penfield and Jasper's (1954) secondary sensory areas. The areas these investigators describe seem to be regions within the limbic system (insula and cingulate gyrus). We have suggested that the limbic cortex serves the appraisal of sense impressions. Here, the only regional organization seems to be provided by the connection of the insula with the lateral part of the somesthetic region (serving sensations from tongue, mouth, face, hands, and arms), and of the posterior cingulate gyrus with the dorsal and medial

In the somesthetic cortex (including the taste area) and the olfactory cortex, the area for recognition has not been determined as yet. However, since area 2 is known to be connected with medullary reticular inhibitory pathways (Glees, 1944), it seems likely that area 3 in the depth of the central sulcus is the primary and area 1 in the postcentral gyrus, the secondary somesthetic area. Electric stimulation of the latter area produces sensations of "tingling," "numbness," or "feelings of electricity," which suggests that such stimulation reactivates earlier patterns of that nature, so that these sensations are recognized.

Such recognition, be it understood, is not a recognition of words or objects, their meaning or use, but a simple experience of familiarity; of having seen, heard, smelled, touched, or felt this before. This recognition may be spurious, as in the "déjà vu" experience, when we seem to recognize something we have never encountered before. In this case, there seems to be some defect in neural conduction; the impulses seem to activate a pattern in the area for recognition instead of establishing a new pattern. This notion seems the more likely as "déjà vu" experiences occur only occasionally in healthy young people but are had more often during old age and are a frequent and conspicuous symptom in mental disease (see Bellak, 1958). An experience of familiarity may be genuine, yet we are unable to recall anything about the object or situation. We may really have seen this face before or heard this particular musical phrase, but are unable to recall when and in what connection, or whether we have liked or disliked it. This is a defect in recall rather than in recognition.

Recall. Merely recognizing something as having experienced it before gives us no clue as to what should be done about it here and now. We must recall how it has affected us in the past, what we did about it, and whether our action was successful or not. In the same way, if we see

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part of the somesthetic cortex (mediating sensations from trunk, legs, and genitals). This explains why neither group of investigators has been able to find an exact regional localization in their "secondary sensory" areas.

something we have never seen before, we must recall similar objects and situations, their effect on us, and our successful or unsuccessful attempts to deal with them.

To recall anything at all, we must produce a visual, auditory, or other image of it. This requires a careful focusing on the exact pattern experienced before. It can be done spontaneously (as, for instance, when the sight of a picture recalls the name of a friend whose favorite it is), or deliberately (as, for instance, when we try to remember the name of an acquaintance). In the first case, the focusing is done for us; in the second, we must find some way of doing it. The work of learning and memorizing consists not in "stamping in" a sensory impression but in learning to refocus on it and so recall it.

To recall a particular experience, it must be possible to reactivate specific registered patterns in the right order. Sensory impressions are received in a definite temporal sequence. An event is experienced as beginning, continuing, and finally coming to an end. To recall it correctly, the registered sense impressions must be activated in the same order, or it might happen that we remember the action in reverse. Instead of seeing the batter hitting the ball and the ball landing in the outfield, we might see the ball rising from the field and coming toward the batter. Whether recall is spontaneous or deliberate, it is selective. We do not recall indiscriminately everything we have experienced before. When the child sees the physician with a hypodermic needle, he recalls this same man holding the same instrument. He recalls what the man did with it and what pain it brought. In the same way, we want to call a particular name to mind, out of the thousands of names we have heard before. Recall seems to bring back *relevant* memory images and do so in the *original order*.

To recall memories in the right order, there must be a circuit from cortical and limbic sensory areas that connects with the thalamic sensory nuclei in such a way that it accompanies the visual or other sensory projection back to the sensory projection area. To recall relevant memories, this circuit must connect with a cortical structure that allows selective activation of memory engrams. The *hippocampus* is a primitive type of cortex and has the necessary connections from sensory and limbic cortex (see Ch. 2) that would make it possible to relay sensory impulses (after perception and appraisal) to midbrain and thalamic nuclei. Its large efferent pathway, the pre- and postcommissural fornix, connects with the septal area and sends relays to the midbrain reticular formation (Nauta, 1956); and this midbrain area is connected with the sensory thalamic nuclei and the sensory, association, and limbic cortex (cf. French *et al.*, 1953). We are suggesting that this circuit mediates the recall of relevant memory images in every sense modality, and at the same time serves to recall whether we have liked or disliked the remembered objects or scenes (see Fig. 5).

To have any memories to recall, sense impressions must be registered at the time they are experienced. We believe that the sensory projections carry impulses not only to the primary sensory areas but also, and simultaneously, to the secondary areas (for recognition), to the association areas (for recall), and the limbic areas (for evaluation). The connections to the limbic areas seem to travel via the brain stem reticular formation and the medial thalamus, with the sole exception of the olfactory system. In addition, there seem to be connections from the sensory areas to the adjoining limbic areas (see Pribram and MacLean, 1953). That there is such a registration of sensory impressions via sensory thalamic nuclei to the association and limbic cortex is shown by several findings. Bagshaw and Pribram (1953) reported that the ventroposterolateral nucleus (the thalamic relay station for somesthetic impulses) degenerated after lesions of the parietal operculum and insula; and Diamond and associates (1958) found that the medial geniculate nucleus (the thalamic relay station of the auditory pathway) degenerated only when the auditory cortex I and II as well as the auditory association area and the posterior insula were removed on that side.

If the recall of memories requires a circuit that repeats the path of the original registration of sense impressions, as we suggest, we would expect that the sensory thalamic nuclei would be excited in different parts, accordingly as we either experience sense impressions or recall memories. A recent report suggests that this might actually be so, at least in the visual system. De Valois and associates (1958) found by multiple cell recordings from each of the six layers of the lateral geniculate nucleus (three layers from each eye) that the three pairs of layers responded differently to light. The dorsal pair (layers 6 and 5) emitted a burst of electrical activity which continued at a slightly reduced rate as long as the light did. The most ventral part (magnocellular layers 2 and 1) showed at first a brief burst, and then a marked inhibition of electrical activity during light, resuming their spontaneous activity as soon as the light stopped. The middle pair (layers 4 and 3) showed increased activity at the beginning of

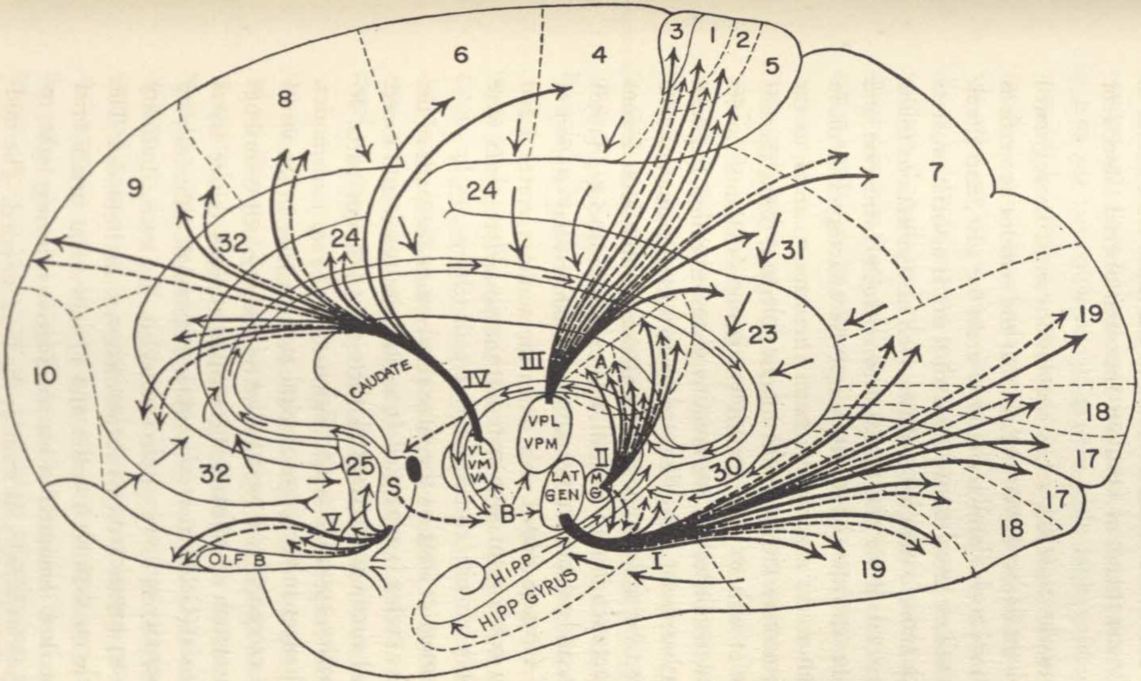

FIGURE 5. THE MEMORY CIRCUIT—RECEPTION AND REGISTRATION, RECOGNITION AND RECALL

Sensory impulses travel via thalamic sensory nuclei to cortical sensory and association areas, mediating sensory experience and later recognition. Associated impulses are relayed to limbic areas, mediating appraisal. This appraisal of sense experience initiates spontaneous recall of similar things and situations, mediated via hippocampus, thalamic sensory nuclei, and the various association and limbic areas. Motor impulses travel via ventral thalamic nuclei to frontal areas.

——— Reception and registration. – – – – Recall. Arrows indicate the direction of conduction. Short arrows indicate the connections for appraisal and recall.

I visual system. II auditory system. III somesthetic system (including taste). IV motor system. V olfactory system. Arabic numerals represent Brodmann areas. A cortical auditory area. B brain stem. HIPP hippocampus. LAT GEN lateral geniculate nucleus. MG medial geniculate nucleus. OLF B olfactory bulb. S septal area. VA anterior ventral nucleus. VM ventromedial nucleus. VL ventrolateral nucleus. VPL ventroposterolateral nucleus. VPM ventroposteromedial nucleus.

Source: Outline of brain structures redrawn from W. J. S. Krieg. 1957. Brain mechanisms in diachrome. 2d ed. Brain Books, Evanston, Ill. Pathways specially drawn.

the light, though it was not as pronounced as in the two dorsal layers, but there was also increased activity as soon as the light stopped. We would suggest that the *dorsal layers* mediate the relay to the primary visual areas: we have a visual sensation as long as there is light. The *ventral layers* seem to mediate the experience of familiarity, of having seen this before: we recognize something right after we see it and when we do, that is the end of this particular experience. The spontaneous activity of this pair of layers between light flashes apparently serves the recognition of things either seen or remembered. Finally, the *middle pair* seems to serve the registration and recall of visual memories: when we see anything at all, we compare it with earlier visual impressions, and when there is nothing to see any longer, a vivid memory image remains. The spontaneous activity of this pair seems to represent the minimal activation of visual memories between stimulation.

This suggested memory circuit makes it possible to solve some persistent problems, of which we will mention only one. Cragg and Temperley have pointed out that every explanation of memory that postulates traces in the brain has so far been defeated by two persistent paradoxes: "The engram is distributed throughout the cortex, yet is not dependent on direct connexions between distant cortical areas; the engram can be established very rapidly, and yet can endure gross changes in the physiological state of the cortex" (1955, p. 306).

Our explanation resolves the two paradoxes easily. First, there is no intracortical memory circuit; consequently crosshatching of the cortex cannot destroy memory as long as the subcortical connections are intact. However, when the cortex is separated from its subcortical connections, or the hippocampal circuit is interrupted, there are gross memory losses (Cragg and Temperley, 1955; Delay and Brion, 1954).*

Second, gross changes in the physiological state of the cortex do not interfere with the *reception and registration* of sensory impressions because the sensory system is resistant to such physiological changes as anesthesia and anoxia (Gellhorn *et al.,* 1954), and registration occurs at the same time as sensory reception and is part of it. But such changes do interfere with *sensory experience* and the *activation of memories.* Sensory experience, we believe, depends on the appraisal of sense impressions, mediated by the limbic system; and the activation of memories in recall, on the hippocampal circuit. Both these connections represent a multisynaptic pathway which is extremely susceptible to a variety of physiological changes (see Brazier, 1954). During sleep, anesthesia, and anoxia, when these pathways are depressed, we are not aware of sense impressions, nor are we able to remember them or initiate action.

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* For our purposes it is immaterial what the engram eventually turns out to be or what is the mechanism of registration. At the present time, an analogy with the magnetic alignment of electrons on a recording tape seems more adequate than the analogy with engraving upon which the term engram is based.

Imagination. Even after an object has been correctly identified and its effect on us has been recalled, we would not be moved to action unless we expected that the same or a similar effect would follow. Such expectation is automatic if this is an exact repetition of an earlier experience. But if what we see or hear now, merely resembles something familiar, we have to guess at its probable effect, taking the changed circumstances into account. This guess may require reasoning, but in many cases it merely requires a rearrangement of memory images, that is, imagination.

Imagination is active every time we have a new sensory experience that demands a different action. There must be some circuit that allows a shuffling of memory images, a rearrangement and new construction, such as occurs in planning, but also in dreaming and imagining. This circuit must include some structure that can be excited by any object that is perceived and appraised, but also by a physiological appetite (for instance, hunger, sexual tension, etc.; see Ch. 6). If our conception of brain function is correct, this would mean that such a structure would have to receive afferents from sensory and limbic areas as well as from hypothalamic nuclei, and would have to project to association and limbic areas. Moreover, since imagined scenes are appraised for action just as real scenes are, there should be some provision for the innervation of motor nerves.

The *amygdaloid complex* seems to be the structure that could serve imagination, for it has the required connections. It includes several nuclei: the medial, cortical, and central nuclei in the anterior part, the larger basal and lateral nuclei in the posterior part. The phylogenetically older medial, cortical, and central nuclei are connected with the septal area via the diagonal band, and with the olfactory tubercle and orbital cortex via the lateral olfactory tract (Kaada *et al.,* 1954; Gloor, 1955; see Fig. 6). Something smelled that is recognized and appraised as good (via orbital cortex and subcallosal gyrus) can induce, say, the visual image of meat or banana, by relaying nervous impulses to the diagonal band and amygdala, and from there to the visual association cortex. The basal and lateral amygdaloid nuclei are more prominent in primates than in other mammals, and

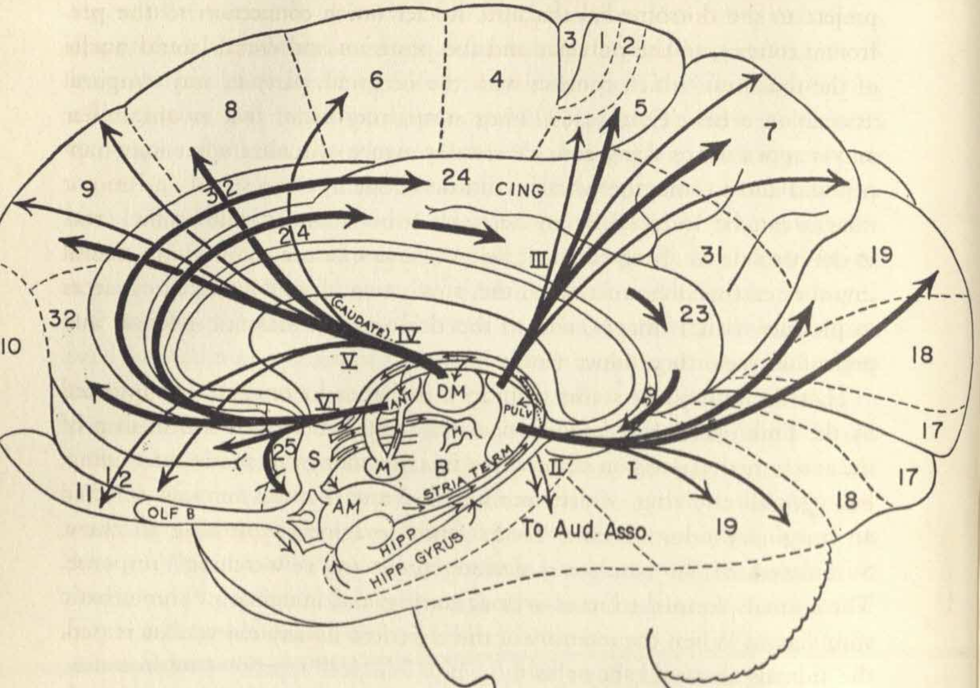

FIGURE 6. THE IMAGINATION CIRCUIT AND THE RECALL OF SUCCESS AND FAILURE

Identification of perceived object (via memory circuit) and its appraisal (via limbic areas) result in imagining possible consequences (relays via amygdala to pulvinar and visual, auditory, somesthetic association areas; to dorsomedial thalamic nucleus and olfactory association area). This gives rise to imagining possible actions (relays via amygdala to dorsomedial thalamic nucleus and frontal motor association area). When an action is contemplated, its success or failure in the past is recalled (via the hippocampus-fornix circuit to the mammillary body, anterior thalamic nucleus, and cingulate gyrus) before it is actually chosen.

I visual imagination. II auditory imagination. III somesthetic imagination (including taste). IV motor imagination. V olfactory imagination. VI recall of success or failure. AM amygdala. ANT anterior thalamic nucleus. B brain stem. CING cingulate gyrus. DM dorsomedial thalamic nucleus. H habenula. HIPP hippocampus. M mammillary body. OLF olfactory bulb. PULV pulvinar. S septal area. STRIA TERM stria terminalis.

Source: Outline of brain structures redrawn from W. J. S. Krieg. 1957. Brain mechanisms in diachrome. 2d ed. Brain Books, Evanston, Ill. Pathways specially drawn.

project to the dorsomedial thalamic nuclei (with connection to the prefrontal cortex), to the pulvinar and the posterior and dorsal lateral nuclei of the thalamus which connect with the occipital, parietal, and temporal association areas (Fox, 1949). Thus something heard that is unfamiliar and is appraised as dangerous can lead a man to visualize what may happen and also to imagine what he can do about it; these visual and motor images can be induced by connections from auditory and limbic areas to the basolateral amygdaloid nuclei. Via intranuclear connections, neural impulses can next be relayed to the pulvinar and visual association areas to produce visual images, and to the dorsomedial thalamic nucleus and prefrontal areas to produce motor images.

That the amygdala seems to induce impulses to imagine is suggested by the findings of Kaada and associates (1954). Electrical stimulation of the anteromedial division of the amygdala in waking cats produced sniffing, licking, and chewing, which seems to indicate that the animals detected an imaginary odor or taste, or that they imagined something to chew. Stimulation of the basolateral division produced a "searching" response. The animals seemed to react to imaginary visual, auditory, or somesthetic stimulation. When the intensity of the electrical stimulation was increased, the animals showed rage or fear.

The motor innervation we have postulated as a result of such impulses to imagine seems to be served by the efferent connections of the amygdala (stria terminalis, connecting with the stria medullaris and the medial forebrain bundle) to the brain stem and spinal motor pathways (see Fig. 6). Gloor (1955) reported that short latency responses appeared in the stria terminalis, septal area, anterior hypothalamus, and tegmentum, on stimulation of the anteromedial division of the amygdala. Apparently, the basolateral division sends impulses to the same areas via the anteromedial division.

That the amygdaloid complex is actually excited during imagination is shown by the report of Lesse *et al.* (1955), who found distinct changes in the electrical activity of the amygdala and rostral hippocampus every time a patient with implanted electrodes vividly imagined or thought about something. Visual, auditory, and olfactory images all produced similarly increased electrical activity.

In summary, we propose that the impulse to RECALL ***what has been experienced*** and ***whether it was liked or disliked*** is mediated by the hippocampal circuit, while the impulse to IMAGINE ***what sensory experience will*** *follow* and *what can be done about it* is mediated by a circuit including the amygdala. Since there is a direct connection from limbic cortex to both hippocampus and amygdala (Green and Adey, 1956), an object perceived and appraised will not only recall relevant memories but will also induce sensory and motor images. Though two different circuits seem to be involved in recall and imagination, it is obvious that the two functions usually act together, at least during waking. We imagine what we cannot recall, or at least, fill in by imagination what we cannot accurately remember. What we recognize and recall usually requires some action for which imagination provides a plan. And what we expect to happen or do must be evaluated and compared with earlier events and earlier action, which requires the hippocampal circuit.

Experimental evidence. Our hypothesis can explain the voluminous and often contradictory evidence from experimental stimulation and destruction of the amygdala. For instance, Brady and his coworkers (1954) destroyed the amygdala of cats on both sides. They found that these animals needed many more trials than intact cats to learn escaping from a click that announced an electric shock. But if they had acquired this habit before the operation, they did not lose it. Since destruction of the amygdala does not abolish an established habit, it cannot affect recall, a conclusion that agrees with results in human beings after such an operation (see Scoville and Milner, 1957).

But to *establish* a habit requires imagination as well as memory. To escape on *feeling an electric shock* merely needs an intuitive appraisal that this is harmful, which results in pain and an immediate impulse to escape. But to run on *hearing the click* depends on remembering that the click was followed by a painful shock, and on anticipating (i.e., imagining) shock and pain. To form an appraisal for appropriate action also requires a motor image of what to do to escape. Without amygdalae, neither sensory nor motor imagination seems to be possible.

Brady's cats without amygdalae did not show fear as did normal animals on hearing the click. Fear can only be felt when some harm is anticipated. Without these nuclei, the electric shock apparently could no longer be anticipated. Accordingly, there could be no fear nor the usual autonomic changes that go with fear (crying, defecation, urination, etc.). In contrast, cats with intact amygdalae but with a lesion in the ventral part of the internal capsule (interrupting the projection from the ventral and dorsomedial thalamic nuclei to the prefrontal area) showed fear and distress on hearing the click, but could neither remember nor relearn to escape before the shock. Since the amygdalae were intact, these animals could anticipate the shock and feel fear. They showed the usual distress, but they could no longer imagine *what to do* to avoid it because the motor association area in the prefrontal lobe was disconnected from the amygdala on both sides. For this reason, the cats mentioned by Pechtel *et al.* (see Ch. 2, Evaluation of Smell) lost a conditioned olfactory discrimination habit after destruction of the dorsomedial thalamic nuclei. They could no longer imagine *what to do* when confronted with various odors. Animals without amygdalae can *retain* a simple, well-established habit as long as the hippocampal (memory) circuit is intact. They can relearn, though slowly, because the memory of shock and pain is gradually recalled together with the memory of what they had done to escape it. They seem to go through the same motions automatically, without fear and without the autonomic changes induced by fear. But they are completely unable to go through a complicated sequence of actions because they can no longer imagine what is the next thing to do.

Morrell and associates (1956) showed that memory and imagination are impaired when amygdala and hippocampus are functioning abnormally. After injection of alumina cream into the amygdaloid nuclei of one side, monkeys developed major and minor epileptic attacks. Simple conditioning to a tone, to a minimal electrical shock, or to a change in background lighting became very difficult, and differential conditioning was altogether impossible. In these experiments, the unconditioned stimulus was a flickering red light and the unconditioned response a change in electrical cortical activity to match the flicker frequency. The conditioned response, the cortical change to a tone announcing the flicker, also requires memory (that the tone was followed by flicker) and imagination (the expected malaise). The injected alumina cream spread to hippocampus, temporal and frontal lobe, operculum and insula, the anterior thalamic nucleus, basal ganglia and internal capsule. This explains why memory as well as imagination was disturbed. It was very difficult to remove this large steadily discharging area; but when it was done, the conditioned reflex improved, apparently because the intact side could now function without interference from the irritative focus.*

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* That disturbance of function in the amygdala and hippocampus can actually distort perception is shown by the experience of patients with psychomotor epilepsy who have a wave and spike focus in the EEG over the anterior temporal lobe. During an attack, the patient may see things larger or smaller, may feel

In recent years, there have been several reports of remarkable changes in the behavior of animals after bilateral ablation of the amygdala and prepyriform cortex, comparable to some of the changes reported by Klüver and Bucy after ablation of the temporal lobe. Schreiner and Kling (1953), for instance, reported that agoutis, monkeys, cats, and lynxes became hyperactive and restless; the animals picked up everything indiscriminately, repeatedly sniffing, licking and pawing their cages and everything in them. They also showed increased sexual activity. After the first preoperative month, females accepted males even when not in heat, and males attempted copulation continuously, not only with females but with males and even with animals of other species. When operated males were castrated, this behavior disappeared within a week. If castrated before bilateral removal of the amygdala, this hyperactivity never developed.

Apparently, the amygdaloid nuclei are necessary for an approach to the appropriate mate. Without them, the animals do not seem to form an image of mate or mating and indulge in random sexual activity as soon as the gradually rising hormone level arouses an impulse to mate. In normal animals, the impulse seems to induce an image of the right mate which directs their approach and images of mating which direct their actions (see Ch. 6).

There are conflicting reports that such animals are either less aggressive (Schreiner and Kling, 1953; Bard, 1953) or more aggressive (Bard and Mountcastle, 1947). The operated animals seem to show no fear of human beings or animals. Cats may investigate a mouse, sniff at it and lick it, but do not catch or kill it. Rats will retrieve and lick mice instead of killing them as do normal rats (Bard, 1953). According to Schreiner and Kling, most of the animals became docile after the operation and showed increased pleasure on petting. Their behavior occasionally resembled sex play, particularly after sexual hyperactivity had developed. Two females, however, became increasingly irritable as their sexual activity increased, and began to bite and claw when anyone approached them. This reaction also occurred when two amygdalectomized cats were spayed and after a week or two of perfect docility were given sex hormone (0.5 mg. diethyl stilbestrol) daily for two weeks or more. After several days, they became less friendly and attacked on slight provocation, though they could be

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that they are strange, remote, or threatening. When the discharging focus is removed by ablation of the anterior temporal lobe, these feelings disappear together with the psychomotor seizure (Penfield and Erickson, 1941; Bailey *et al.*, 1953).

handled if gently approached. When the injections were stopped, they became docile again.

Animals without amygdalae (and thus without spontaneous imagery) no longer know what to expect; thus any abrupt interference with their activity may arouse anger, no matter how well they know the attendant. According to Pechtel (1955), an amygdalectomized animal becomes savage if handled roughly. But if it is approached slowly and handled particularly gently such savageness will not develop. Apparently, when increased hormone secretion or the injection of sex hormone results in an urge to activity without the image of mate or mating to give it direction, there is an exaggerated reaction to interference. When a slow and gentle approach demonstrates to such animals that no harm is coming to them, they will remain quiet. Their attack is prompted neither by fear nor by viciousness; it is merely a hasty response to an unforeseen movement.

The experiments of Lesse and associates and Morrell and his coworkers have shown that the hippocampus is activated whenever the amygdaloid nuclei are stimulated. When not too intense, the electrical activity may be confined to these two structures (Lesse *et al.*, 1955). When stronger, it may induce overt action (Kaada *et al.*, 1953; Penfield and Jasper, 1954), probably via the hippocampus and action circuit; and when excessive, may result in convulsions (Morrell *et al.*, 1956; Creutzfeldt and Meyer-Mickeleit, 1953). Penfield and Jasper, for instance, have reported that a patient whose epileptic attacks started from a focus in or near the amygdala sometimes had an epileptic seizure when he tried to recall a name or other memory. In this case, trying to remember seems to have initiated seizure activity in the hippocampus, which eventually spread to the motor cortex.

When either the amygdalar or the hippocampal circuit is interrupted, we would expect an interference with actions that depend on imagination and memory. The interruption of the memory circuit seems to be the cause of the memory defects after brain damage reported by various investigators. Memory disturbances have been found after temporal lobe tumors (Simma, 1955) which probably affected the hippocampus, after third ventricle tumors (Williams and Pennybacker, 1954), after lesions in the midbrain reticular formation (Hernández-Peón *et al.*, 1956), and after damage to hippocampus and mammillary bodies (Ule, 1951; Grünthal, 1947).

The reports of Dott (1938) and of Garcia Bengochea and associates (1954; see Ch. 2, Memory Traces and Their Location) that cutting the descending fornix columns produces no memory defects can be explained by the fact that these tracts are not the only efferents from the hippocampus. According to Nauta (1956), the hippocampus projects via both pre- and postcommissural fornix fibers to the septal cell groups, the preoptic area, the dorsal hypothalamic area, and the periventricular hypothalamic zone, and to the infundibular region; postcommissural fibers go to the mammillary bodies via the descending fornix columns, and only these were cut by Dott and Garcia Bengochea *et al.* Since there are connections from the septal nuclei to the midbrain, it seems likely that this alternative pathway is sufficient for recall when the connection to the mammillary bodies is broken. But with large lesions of the septal area, damaging pre- as well as postcommissural fornix fibers, the memory circuit can probably not function any longer. According to Brady and Nauta (1953; 1955) rats with such lesions were difficult to handle, showed violent startle to sound, and fought continuously among themselves. These reactions can be explained as memory defects. Like Klüver and Bucy's monkeys, the rats apparently could not remember how things had affected them in the past. But their imagination circuit was intact and they could (and evidently did) expect harm from almost everything. They showed as much fear on hearing a sound that announced an electric shock as did normal animals, but probably not for the same reason. Right after the operation, half the group did not show the conditioned fear response, apparently because they could not remember what the sound meant. But they did apparently expect harm from every loud sound and so reacted to the sound stimulus with fear also. Very soon the whole group reacted with fear whenever they heard the click.

The importance of the hippocampus for the organism can be gauged from the fact that it is among the first structures that show electrical activity when the hamster comes out of hibernation (Chatfield and Lyman, 1954). To orient himself and look for food, the animal must remember where food is to be found and what has been good to eat or what should be avoided.

The interruption of the imagination circuit seems to be responsible for the loss of "memory" mentioned by Scoville (1954) after bilateral ablation of uncus and amygdala. His patients were unable to find their way around the hospital or even their own house. This could be a loss of imagination rather than a loss of memory. Human beings are not used to following a motor habit blindly. They try to orient themselves in their environment, and become confused when they can no longer imagine what to expect or what is to be done. Animals do not lose a well-established motor habit after ablation of both amygdalae, as we have seen. That the loss of the amygdala on both sides does not produce a memory loss is shown by the report that bilateral ablation of the hippocampus and hippocampal gyrus, or of the lateral temporal cortex, results in loss of "recent memory," while bilateral ablation of the amygdala does not (Milner and Penfield, 1955; Scoville and Milner, 1957). This seems to be a loss of visual and auditory memory, which are the modalities most frequently employed; they seem to be mediated by the temporal cortex (auditory memory) and the pathways through the temporal lobe to the occipital association area (visual memory). Early memories are retained, probably because they have been recalled and recounted over and over and have become speech (motor) memories, which can be activated via the remaining portion of the hippocampus.

THE CHOICE OF ACTION

When we have identified what we perceive, when we have recalled its earlier effects, when we expect a similar effect and have imagined possible ways of meeting it, we finally choose an action and get ready to carry it out. But even the first appraisal of sensation, mediated by the medial thalamus, implies potential action and seems to initiate relays to motor pathways. We know from experience that a sudden sound in the stillness of night will startle us (appraisal via the medial thalamus) and make us tense apprehensively, long before we have identified the source of the sound or have decided how to deal with it. Action impulses occur as soon as anything is evaluated at all, but they are ordinarily held in check until the situation has been identified and appraised for appropriate action.

The intention to act in a particular way must be appraised, in turn, with the help of memories of earlier success and the pleasure connected with it. This appraisal, we believe, is mediated by a circuit from the hippocampus via the fornix to the mammillary bodies and from there via the mammillothalamic tract to the anterior thalamic nucleus and the cingulate gyrus which seem to serve the appraisal of movements and somatic sensations. The cingulum connects these areas with the hippocampus (see Fig. 6). When such appraisal of planned actions is impossible, we would expect considerable difficulty in learning an action pattern. Actually, the interruption of the cingulum has resulted in a serious deficit in maze learning in rats (Thomas and Otis, 1958), depending on the extent of the lesion.

When action is finally decided upon, the hippocampus seems to send impulses via the fornix and mammillary bodies to the midbrain, cerebellum, and frontal lobe to connect with cells in the motor cortex. The hippocampus seems to transform what could be called sensory impulses (i.e., impulses relayed from sensory, association, and limbic areas) into motor impulses (i.e., impulses relayed to structures that have a motor function). Thomalske and associates (1957) have mentioned that a change in the electrical activity of the hippocampus (recorded from implanted electrodes in waking animals) occurs just before the animal begins to move. They suggested that this structure may signal the intention to move, which is in line with our own hypothesis. The architecture of the hippocampus also seems to support such an interpretation. There is a strip of granular cortex that has the same cell structure as the primary sensory cortex, and a strip of agranular cortex that has the same cell structure as the motor cortex (Von Economo, 1929).

Sometimes there is a period of hesitation or deliberation before action. The situation may have conflicting aspects, some pleasant, some unpleasant. Or the effect of various possible actions has to be estimated. In all these cases, there seem to be repeated rounds of appraisal which allow recalling more and different aspects of the situation, and making different plans for action. All these appraisals, even those that do not lead to action, seem to arouse action impulses that are experienced as conflicting emotions and expressed in face, posture, and muscular tension.

Such a process of repeated appraisal would account for the occurrence of rhythmic after-discharges in the hippocampus on sensory and electrical stimulation. Andy and Akert (1955) report, for instance, that the electrical after-discharge starts with a slow rhythm, abruptly shifts to a fast rhythm of high amplitude, and may go on for as long as two minutes until it stops abruptly. And Kaada (1951) found that such seizure activity in the hippocampus has a tendency to spread over the limbic lobe, but is not always confined to it. We suggest that these after-discharges accompany the whole process of recall and appraisal, which will go on until some action is chosen, or until man or animal decides that no action need be taken, when it will stop abruptly.

According to Green and Arduini (1954), sensory stimulation results in a primary electrical potential at arrival of neural impulses in the sensory cortex. This is later followed by a diffuse wave. A primary response to sensory stimulation is also observed in the hippocampus, followed after some delay by a secondary response in this structure, the rhythmic after-discharge, which lasts for some time. The primary response would correspond to the impulses received in sensory and association areas and limbic cortex. The secondary response, much longer in latency, seems to appear in these cortical areas as soon as neural impulses have been relayed there from the hippocampus, to activate memory traces. It seems to appear in the hippocampus when the intention to act is to be appraised (via the cingulate gyrus) and neural impulses are relayed back to the hippocampus to initiate an impulse to action. The so-called "seizure activity" or "after-discharge" in the hippocampus seems to represent the activation of memory and appraisal until a definite action is chosen.

Liberson and Akert (1955) found that the hippocampal after-discharges spread also to the opposite hippocampus and to other cortical areas (mostly central and posterior rather than frontal), which is exactly what we would expect if these after-discharges represented the recall of relevant memories. In the subcortex, electrical activity was found in the thalamus, striatum, amygdala, and hypothalamus. On repetitive stimulation of sensory nerves (which is more like natural sensory experience than is single shock stimulation), there is a similar high amplitude secondary wave which diffuses neural impulses widely over the cortex. These changes in the electrical activity of the cortex do not occur if the hippocampus is removed. Instead, there is a serious reduction of cortical potentials, with wave spindles and periods of complete inactivity, a picture resembling the EEG in deep narcosis (Green and Arduini, 1954). We may infer that it is hippocampal excitation which sets off these diffuse discharges and is indirectly responsible for normal electrical activity in the cortex.

According to our conception, the animal's psychological activity would be seriously disturbed when the hippocampus was removed. Of course, in practice the removal is never complete; for the hippocampal rudiment, arching over the corpus callosum, remains connected with the fornix and allows the recall of motor and touch memories, at least to some extent. This explains, for instance, why the patient reported by Terzian and Dalle Ore (see Ch. 2) could say a few sentences that were connected with his immediate needs (food, cigarettes, etc.), yet could not carry on a conversation, for without memory he could not understand what was said to him. If the hippocampal system could be totally removed without inflicting any other damage, there would be no meaningful perception, because recall would no longer be possible, and no goal-directed action. With the amygdala intact, imagination would be stimulated by each sense experience; there would be great fearfulness, for nothing would work as expected, once memory had been excluded. The same effect is observed after large lesions in the septal area, which destroy both the pre- and postcommissural fornix (cf. Brady and Nauta, 1953, 1955).

ORGANIZATION OF THE ACTION PATTERN

We have now seen that an object or a situation is perceived, appraised, and liked or disliked; and that this liking or disliking arouses a tendency to approach or withdraw, to deal with this thing in some particular way. Hence we have defined emotion as the felt tendency toward something appraised as good (and liked) or away from something appraised as bad (and disliked). According to our conception, the limbic system mediates liking or disliking, while the hippocampus seems to initiate the recall of memories, and also the impulse to a particular action. The relay from the hippocampus seems to touch off the whole action pattern of a given emotion, everything that goes into making it fear or anger or love. This would imply that the various action patterns have to be organized and synchronized before they can be relayed to the motor cortex and so lead to actual movement.

We hope to show that these action patterns are organized in the cerebellum and relayed to the frontal lobe (see Fig. 7). While the appraisal for action, as we have described it, implies that a specific action is already decided on when relays start from the hippocampus, the over-all pattern of body and limb movements, of head, eye, or hand movements, complete with endocrine changes and autonomic adjustments, cannot be coordinated in that structure. And it is only when the urge to action is felt all over, when the child strains toward the mother with its body as well as with eyes and arms, that we can say emotion is actually ***experienced*** as an ***action tendency***. Even when the emotion is not strong enough to lead to immediate movement or action, whenever it is felt at all, it is felt in this way: as an urge to a specific kind of action which propels whatever limbs or muscles are necessary toward the thing wanted or away from the thing feared.

The experiencing of emotion as a tendency to a particular action seems

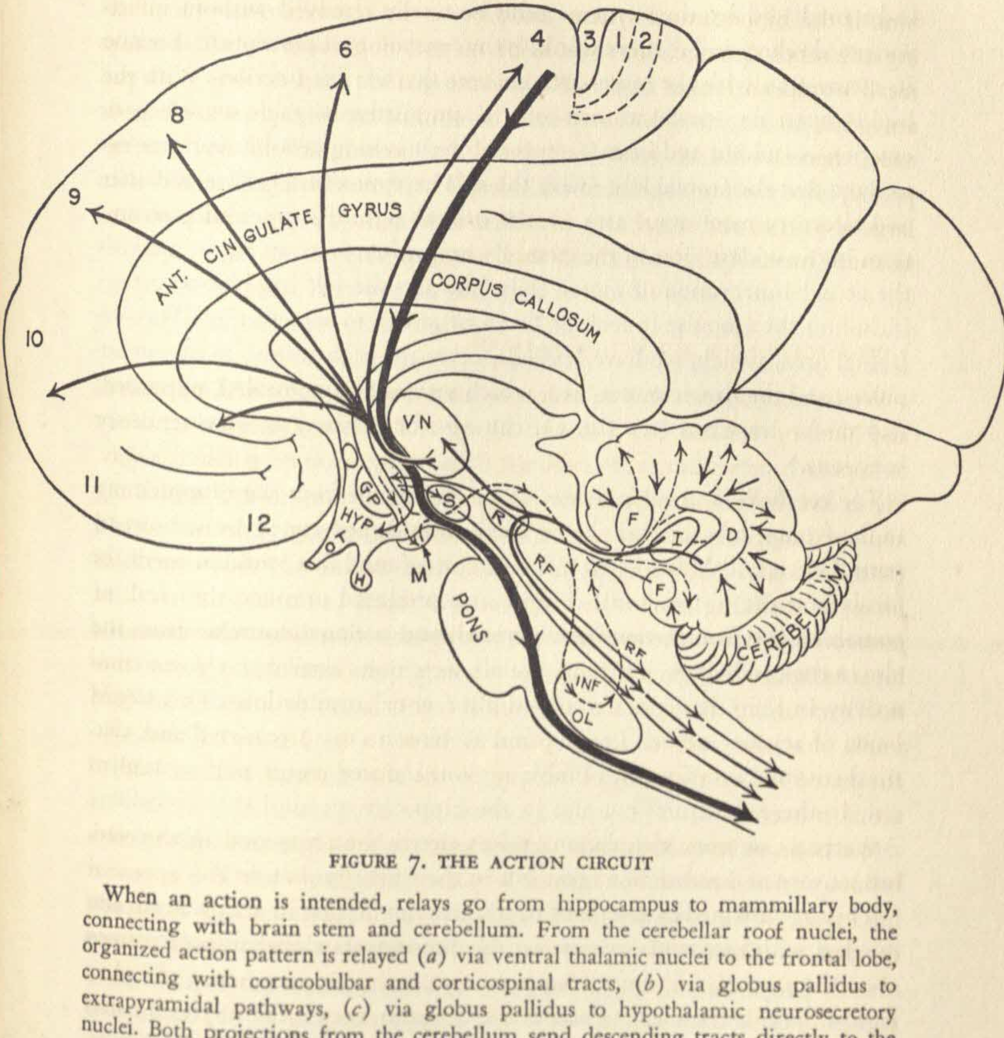

FIGURE 7. THE ACTION CIRCUIT

When an action is intended, relays go from hippocampus to mammillary body, connecting with brain stem and cerebellum. From the cerebellar roof nuclei, the organized action pattern is relayed (*a*) via ventral thalamic nuclei to the frontal lobe, connecting with corticobulbar and corticospinal tracts, (*b*) via globus pallidus to extrapyramidal pathways, (*c*) via globus pallidus to hypothalamic neurosecretory nuclei. Both projections from the cerebellum send descending tracts directly to the spinal cord.

– – – – – Relay from hippocampus to cerebellum. ________ Relay from cerebellum to frontal lobe. ________ Relay from cerebellum to extrapyramidal and hypothalamic effectors. ▬▬▬▬ Corticospinal tract.

D dentate nucleus. F fastigial nucleus. GP globus pallidus. H hypophysis. HYP hypothalamus. I interposited nucleus. INF OL inferior olive. M mammillary body. OT optic tract. R red nucleus. RF brain stem reticular formation. S substantia nigra. VN ventral thalamic nuclei.

Source: Outline of brain structures redrawn from W. J. S. Krieg. 1957. Brain mechanisms in diachrome. 2d ed. Brain Books, Evanston, Ill. Tracts specially drawn.

to be mediated by the structure which receives the fully organized and patterned relay; and that, we think, is the premotor area in the frontal lobe. Its connection with the motor area ensures that the felt urge to action will lead to overt movement as soon as it is appraised as suitable or, when the emotion is sudden and intense, even before it is so appraised. We propose to show that the frontal lobe (with the sole exception of the olfactory bulbs and olfactory association area on the orbital surface) serves motor functions in the widest sense of the term. By motor functions we mean not only the actual innervation of motor cells, but also the felt impulse to action, including the impulse to look or listen or attend to something.* The prefrontal area, which, we have argued, serves the registration of motor impulses, and the limbic motor area, which serves the appraisal of movement and motor impulses (see Ch. 2), can also be counted as serving motor functions.†

The cerebellum as relay station. That the circuit from the hippocampus to the frontal lobe reaches the cerebellum is demonstrated by various reports. Green and Morin (1953), for instance, found that stimulation of the fornix and the hippocampal commissure produced primary electrical potentials in the frontal, temporal, parietal, and occipital cortical areas, and also in the cerebellum. These potentials were quite similar to the electrical activity in these structures recorded after visual stimulation or on stimulation of sensory nerves. Lennox and Robinson (1951) reported that electrical stimulation of various limbic regions induced an after-discharge not only in these structures but also in the hippocampus and the cerebellum. Apparently, sensory stimulation evokes electrical activity not only in various sensory and association areas but in the cerebellum as well.

This is confirmed by Gastaut (1954), who found that in anesthetized cats injected with metrazol, sensory stimulation induces electrical potentials in the sensory cortex, brain stem, cerebellum, and thalamus, and finally evokes spikes in the motor area. We believe that sensations are experienced when impulses arrive in the thalamus and cortex, and that relays from these areas are sent to the cerebellum as soon as man or animal has decided on some action. The direct relays from spinal proprioceptive nerves to the cerebellum, like the direct relays from sensory thalamic nuclei to the tectum and tegmentum, seem merely to induce a generalized muscular tension in preparation for specific action. Since stimulation studies suggest that the cerebellum is a relay station in the circuit from the hippocampus to the frontal lobe, and anatomical findings confirm that the cerebellum projects to the motor and premotor cortex and also to prefrontal areas via ventral thalamic nuclei (Rand, 1954; Cohen *et al.,* 1958), let us see what the role of the cerebellum could be.

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* The *felt* impulse to action should be carefully distinguished from a *neural* impulse. The felt impulse to action is not always intense enough to be called an urge to action, nor attended to sufficiently to be called a desire (for action); hence the term "impulse" is necessary to indicate what we mean. Whenever the context seems ambiguous, we will stipulate explicitly whether a felt impulse or a neural impulse is meant. But we will simply speak of "impulse" whenever it is clear from the context that only one kind of impulse can be meant.

† It is perhaps necessary to mention here that this hypothesis of frontal lobe function is not the result of unsupported speculation but was chosen after exhaustive study of relevant research as the only one that will fit experimental evidence and agree with clinical findings. The hypothesis has to be stated at this point to make it possible for the reader to follow the thread of the argument. Supportive evidence will be given as the argument develops.

The function of the cerebellum. It has been known for many years that the cerebellum is concerned with muscular tone, the facilitation or inhibition of movement, and the maintenance of balance and posture. Together with the basal ganglia, it influences movement and controls muscular coordination. When the cerebellum is removed, there is more interference with normal movement than there is after removal of the cerebral cortex (Chambers and Sprague, 1955). It has usually been assumed that the cerebellum exercises its influence directly on the motor neurons of the spinal cord.

However, Mettler and Orioli (1957) have reported that ataxia cannot be produced by cutting the connection between the cerebellum and the spinal cord (i.e., sectioning the descending limb of the brachium conjunctivum and of the rubrospinal tract). But ataxia follows sectioning of the brachium conjunctivum which projects to the thalamus, premotor, and motor cortex; hence it would seem that the main projection runs from the cerebellum to the frontal lobe. This is supported by the finding of Henneman *et al.* (1952) that stimulation of the cerebellum evokes short latency electrical potentials in the motor cortex, and also by the report of Gastaut (1954), who showed that sensory stimulation, reinforced by metrazol, produces electrical activity in the cerebellum and motor cortex.

When cerebral sensory areas were stimulated, long latency potentials were evoked in the cerebellum, similar to the potentials observed on sensory stimulation. On mapping the cerebellar areas that responded to such stimulation, a precise localization was found. When the "tactile" area in the cerebellum was stimulated in turn, short latency potentials were recorded in the cerebral motor cortex (Henneman *et al.*, 1952) and movements of the corresponding arm or leg could be observed (Hampson *et al.*, 1946, 1952). Thus sensory areas in the cerebral cortex project to definite areas in the cerebellum, but the latter areas project to the motor cortex rather than to the cerebral sensory areas. This would suggest that the cerebellum receives neural impulses from the sensory areas (via the hippocampal system, we believe) and initiates movement by relaying them to the motor cortex. Such an assumption would explain why auditory and visual stimuli evoke electrical potentials in the same cerebellar region that is activated when the face is touched (Snider, 1952; see Fig. 8). Whether something is seen or heard or touches the face, an animal will immediately turn head and eyes in that direction to investigate; and this impulse to action seems to be organized in the cerebellum.

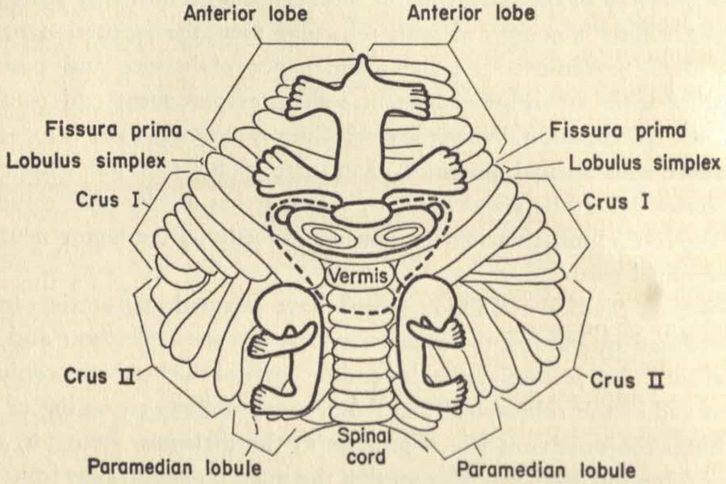

FIGURE 8. CEREBELLAR REPRESENTATION OF THE BODY

When various body surfaces are touched, potentials appear in the dorsal cerebellar cortex and also in both paramedian lobules. Auditory and visual area overlaps the face region.

——— Tactile projection. – – – Visual and auditory projection.

Source: R. S. Snider. Aug. 1, 1950. Recent contributions to our knowledge of the cerebellum. Monthly Res. Rep. Office Naval Res. Diagram specially redrawn.

Snider has found a double "tactile" projection in the cerebellum, one on the dorsal cerebellar cortex (median region of the anterior lobe, lobulus simplex, folium, and vermis) and the other on the ventral surface, in each paramedian lobule (see Fig. 10). It has been suggested that the middle portion of the cerebellum (corresponding to the human vermis) is well developed in animals that depend largely on trunk or symmetrical limb movements, as, for instance, birds and reptiles. With the development of limbs and their eventual use independent of the trunk, the intermediate and lateral cerebellar cortex develop more and more extensively. In man, the cerebellar hemispheres (intermediate and lateral regions) have developed remarkably, corresponding to his extensive use of arms, hands, and fingers (Ranson and Clark, 1953). This would explain the double representation of the body in the cerebellum. The middle region on the dorsal surface apparently initiates movements of the whole body and head in response to what is perceived, though such movements require adjustment of the limbs as well; hence body and limbs are also represented. On the other hand, discrete movements of the limbs always require some adjustment of trunk and head and, in most cases, of every other portion of the body. Accordingly, the limbs are represented on both sides of the cerebellum in the intermediate strip on the ventral surface of both paramedian lobules, but trunk and head are represented there as well.

We would suggest that the cerebellum serves to amplify and organize the neural impulses received from the hippocampus and to coordinate them into a pattern for specific action. In this way, a pattern of movement is set which includes varied and often complicated motions, autonomic changes, and the facial and postural expressions that go with a given emotion. The structure of the cerebellum is admirably fitted for this task: it consists of numerous folia of cortex folded over the medullary white fibers, almost like the leaves of a book. The large majority of all incoming neural impulses go to the cerebellar cortex which has the same structure throughout all its parts (and such homogeneous structure is another argument for the suggested uniform motor function). All impulses leaving the cerebellum start from the roof nuclei (masses of gray cells in the upper part of the white matter) and proceed via the superior cerebellar peduncle (or brachium conjunctivum) and smaller accessory tracts to brain stem and thalamus. A descending limb turns downward at the level of the red nucleus and distributes to the reticular formation in midbrain and medulla oblongata (see Fig. 7).

In the cerebellar cortex, there are sheets of large Purkinje cells which send out many branches to neighboring cells. Thus a neural impulse can induce the discharge of many neurons at once, so-called "avalanche conduction." In this way, impulses from the hippocampus-fornix system can be amplified and distributed to every cerebellar area, controlling the muscles in every limb that are necessary for a given movement. When the cerebellum is removed in animals, uncoordinated (ataxic) movement is still possible, but it is awkward and extremely effortful.

Impulses are relayed to the cerebellum not only via the hippocampus-fornix system but also directly from every area in the cortex via the pons and olive, arriving over the middle cerebellar peduncle (or brachium pontis), while proprioceptive and vestibular impulses are relayed from the spinal cord and medulla oblongata via the inferior cerebellar peduncle (or restiform body). These incoming fibers are in close contact with Purkinje cells that send axons to cerebellar nuclei; and these in turn send relays via the brachium conjunctivum to the midbrain, thalamus, and frontal lobe. The incoming fibers seem to bring information as to what movement is required (hippocampocerebellar fibers), what is the position of the object (corticocerebellar fibers) and what the position of body and limbs (corticocerebellar and spinocerebellar fibers). The outgoing fibers seem to relay impulses to move specific muscles, secrete specific hormones, and innervate the autonomic nerves appropriate to a set action pattern.

Cerebellum and motor pattern. The proprioceptive and vestibular impulses arriving directly from the spinal cord and the medulla oblongata seem to make it possible to compensate immediately for changes of equilibrium during motion and to react automatically to changes of tonus and posture, even without any conscious experience of sensations from muscles or vestibular apparatus.* The neural impulses from the various cortical areas may indicate the position of the body in space at any given moment or of an object in relation to us. In this way, a stream of impulses can be relayed from the cerebellum to the motor area, ever changing with the movement required and made. When this continuous adjustment is impossible, as in various cerebellar lesions, there is an inability to judge distance, hence difficulty in jumping, climbing, and reaching (Rand, 1954; Sprague *et al.*, 1955).

The cerebellum projects to the spinal cord as well as to the motor cortex, via the brain stem reticular formation. This projection seems to be organized in three zones. The entire medial strip of the cerebellar cortex in the anterior lobe sends impulses to the fastigial and vestibular nuclei and from there to the medulla oblongata and brain stem via fibers running in the restiform body and accessory descending tracts. Stimulation of this medial zone seems to produce effects similar to those found on stimulation of the medial reticular formation: slow movements of the entire body or flexion of all four limbs. These movements remain after cutting of the pyramidal tract or destruction of the cerebral motor areas (Sprague *et al.*, 1955).

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* Vestibular impulses arrive in the flocculo-nodular lobe directly from the eighth nerve. Only a few vestibular fibers go to the uvula. The flocculo-nodular lobe is the phylogenetically oldest part of the cerebellum, constant throughout the vertebrate series. It is the only structure that receives and sends fibers exclusively to the vestibular nuclei (Fulton, 1951b).

The most lateral strip of the cerebellar hemisphere projects to the dentate nucleus (lateral zone), while the intermediate strip projects to the interposited nucleus (paravermal or intermediate zone). The intermediate and lateral zones relay impulses to the brain stem via the *brachium conjunctivum,* carrying fibers from the dentate and interposited nuclei, and produce effects similar to those obtained on stimulating the lateral reticular formation. Both lateral and intermediate zones, when stimulated, produce effects that are strikingly similar to those of pyramidal function (Sprague *et al.*, 1955). Since the brachium conjunctivum projects to the ventral thalamus and motor cortex, the inference is justified that the intermediate and lateral zones activate the motor area and produce their effects via the pyramidal tract.

As the descending limb of the brachium conjunctivum and the connecting rubrospinal tract project to the brain stem reticular formation and eventually to the spinal cord, similar movements can be evoked whether the cerebellum or the brain stem reticular system is stimulated. Nor is there any difference in the effect of such stimulation whether decorticated, decerebrated, or normal animals are used.

Spontaneous movement, however, is very different in decorticated and in decerebrated animals. Decorticated cats can walk, but cats decerebrated by a cut between the superior and inferior colliculi in the midbrain can only stand. Apparently, decorticated cats have at their disposal motions that are initiated via thalamic connections. Since the circuit mediating them includes the cerebellum, there is no ataxia; movements can be executed via the red nucleus, brain stem, and spinal cord. When a cut between the superior and inferior colliculi interrupts the connection from cerebellum to red nucleus (producing a decerebrate animal), coordinated walking movements are no longer possible, but electrical stimulation of the brain stem can still activate spinal motor neurons.

Cerebellum and emotional patterns. An emotional pattern always includes a motor pattern activated in so-called "emotional expression." Emotions are reflected in face and posture even if they do not lead to action. When more intense, they urge to specific movements: in anger a man may strike, when afraid of a blow he may shield his face with his arm. We would expect that the emotional pattern, like every action pattern, would be organized and amplified in the cerebellum.

This is confirmed by Sprague and Chambers (1955), who found that destruction of the dentate nucleus of the cerebellum markedly reduces the response to pain in trunk and limbs of the same side, while destruction of the auditory region on both sides greatly reduces startle. In fact, after destruction of the auditory areas in the cerebellum, the animal no longer paid any attention to noises which he had quickly investigated before. These authors point out that earlier investigators had reported increased sensitivity to touch, pain, auditory, and visual stimuli when the cerebellum was stimulated: the animals would howl, cry, snarl, back away, try to escape, cringe, or wildly lick and bite their own body. More recently, Zanchetti and Zoccolini (1954) produced outbursts of sham rage in placid decorticated cats by stimulating the fastigial cerebellar nuclei through implanted electrodes. These emotional outbursts were abolished by coagulation of rostral midbrain structures, apparently because the lesion had interrupted the pathway from thalamus to midbrain and cerebellum. These reports surely suggest that the emotional response to sensory stimuli (that is, the tendency to emotional action) is exaggerated by stimulation of the cerebellum and reduced or abolished when such stimulation is prevented.

THE INITIATION OF ACTION

We have suggested that the choice of action is mediated by the hippocampus which sends neural impulses to the cerebellum, to be amplified and organized into a coordinated pattern. This synchronized impulse pattern is next relayed to the ventral thalamus and frontal lobe. On arrival of impulses in the premotor area, we believe, the urge to action is experienced and can be appraised as suitable or unsuitable when compared with remembered earlier actions. The relay of this impulse pattern from the cerebellum to the frontal lobe will be our next concern.

The most lateral strip of the cerebellar hemisphere projects to the dentate nucleus while the intermediate strip projects to the interposited nucleus in the cerebellum. Both these nuclei send axons in the brachium conjunctivum to various widely distributed end stations. According to Rand (1954) and

Cohen *et al.* (1958), the brachium conjunctivum decussates in a broad sheet, beginning at the border of the pons and isthmus and ending at upper midbrain levels (superior colliculus). These fibers go to tegmental and mesencephalic nuclei until they reach the red nucleus where some of them end, both in small- and large-celled portions. Almost half the fibers go beyond the red nucleus to the field of Forel and distribute to various thalamic nuclei: to the lateral ventral nucleus with connections to the premotor and motor cortex; to the anterior and medial ventral nuclei which project to prefrontal areas and the insula; to the dorsomedial nucleus which also connects with prefrontal and insular cortex (Papez, 1956); and to the centrum medianum and midline nuclei of the thalamus, which project to the anterior cingulate gyrus and diffusely to the whole cortex. Another group of fibers go to the nucleus of the field of Forel and the zona incerta. These connect with the frontal lobes also, probably via the medial forebrain bundle and related tracts in the hypothalamus. Still other fibers end in various oculomotor nuclei.

The medial strip of the cerebellum, on the other hand, sends relays to the fastigial and vestibular nuclei. The axons that originate in these nuclei are collected in the fastigiobulbar tract and the hook bundle, and cross over to become scattered among the fibers of the thalamo-olivary tract and the tegmental gray in isthmus and midbrain, finally ending in the posterior diencephalon and the nucleus of the medial longitudinal fasciculus. The descending fibers distribute to the pons and medulla oblongata, to vestibular nuclei, and to the chief sensory and motor nuclei of the trigeminal nerve (Rand, 1954). The medial longitudinal fasciculus reaches the oculomotor nuclei in the midbrain, and the cranial motor nuclei. Moreover, these fibers from the cerebellum are connected with the hypothalamus and the striatum and globus pallidus, and these, in turn, with the ventral thalamic nuclei and premotor cortex. Stimulation of the medial cerebellar zone induces slow movements of the entire body, which are also produced by stimulation of the premotor cortex, after destruction of the motor cortex. Hence, it seems possible that these movements are brought about via the relay to the globus pallidus which may include the premotor cortex. Very little is known about the effector link of this extrapyramidal pathway.* But the evidence does indicate that lesions of the medial cerebellar zone or the fastigial and vestibular cerebellar nuclei produce deficits in walking, equilibrium, posture, and muscular tone of *the whole body,* while stimulation produces exaggerated tone and movements. On the other hand, stimulation or destruction of the intermediate and lateral zone, or of the dentate and interpositus nucleus, results in interference with fine movements of the *limbs on the same side,* together with mild changes in tone and locomotion (Sprague *et al.,* 1955).

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* There is agreement, however, that caudate and putamen project either directly or via the globus pallidus to the subthalamic nucleus and the substantia nigra. The globus pallidus also sends fibers via the ansa lenticularis, the fasciculus lenticularis, and the subthalamic fasciculus to the subthalamic nucleus, substantia

Emotional expression always engages the whole body. When emotion is intense, it throws the whole body into violent action which makes finely coordinated limb or finger movements impossible. When only a mild emotion is felt (e.g., interest or alertness), the activation is similarly diffuse but will now facilitate action rather than disturb it. Since the whole body is active in emotional expression, the emotional tendency to action should be mediated via the relay from the medial cerebellar zone to the midbrain and the globus pallidus. Actually, electrical stimulation of the fastigial cerebellar nucleus in the medial zone has produced an outburst of rage; and patients with shaking palsy (in which the globus pallidus or its connections with the cerebellum degenerate) have an expressionless, mask-like face and none of the automatic expressive movements of normal people. In contrast, destruction of the dentate nucleus (lateral zone) has reduced the response to painful stimulation in the limbs on the side of the lesion (Sprague and Chambers, 1955), which seems to show that the lateral zone organizes the reaction to pain from local stimulation.

The felt impulse to action. The relays from all the zones connect with the premotor cortex (area 6), some via hypothalamus and striatum, others via the thalamus. We are suggesting that the premotor area mediates the experience of an urge to action which includes movements of the whole body and also discrete movements of one or both arms and hands.* In this area, both types of movement, mediated by different pathways, seem to be combined and experienced as an urge to coordinated action. When

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nigra, red nucleus, and the tegmentum, connecting with rubrospinal, rubrobulbar and tegmentospinal tracts (Peele, 1954; Showers, 1958). We believe that this is the extrapyramidal effector pathway and that other efferent connections from the basal ganglia have a different function.

* If the relays to the premotor cortex mediate an impulse to action, the relays from the premotor to the motor cortex may prime the motor cells for action while the direct relays from the ventral nucleus of the thalamus may activate them. Lashley points out that "there are many bits of evidence which suggest that a neuron must be excited by several or many axon terminations in order to be fired" (Lashley, 1952, p. 544).

area 6 is stimulated electrically, contralateral movements are evoked, very similar to those evoked from the corresponding area of the motor cortex, though they are less discrete. These movements have a longer latency than those evoked by electrical stimulation of the motor cortex, with which the premotor cortex is connected. They seem to be produced via area 4, although the extrapyramidal pathways may have a share in them, providing background adjustments. At any rate, when the motor cortex is destroyed and the premotor cortex is then stimulated, there is a slow movement of the whole body while head and eyes are turned to the opposite side, similar to the slow movement evoked by stimulation of the medial cerebellar zone. Destruction of the pyramids does not interfere with these movements, but undercutting of area 6 abolishes them (Fulton, 1951b).

Stimulation of the lower portion of area 6 on the level of the motor representation of mouth, lips, tongue, and glottis, has evoked chewing, swallowing, smacking of lips, and grunting. Once started, these movements, like the slow body movements evoked from more dorsal points, may persist for some time. When area 6 is isolated from the motor cortex by a cut, this response persists; hence these movements also seem to be carried out via the same extrapyramidal pathway.

From area 8 (the so-called "frontal eye field," which has a cortical structure almost identical with the premotor area) electrical stimulation produces movement of both eyes in the same direction, either left or right, up or down. This seems to indicate that eye movements also may be part of the total body movement, in which case they are mediated over an extrapyramidal route; but there may also be a deliberate looking, mediated via the motor cortex and the corticobulbar fibers. According to our conception, the relay from the cerebellum would prime the motor nuclei of the eye and other cranial motor nerves for action, while the two effector pathways, the cortico-bulbar tract and the extrapyramidal fibers from area 6 and globus pallidus, would activate them.

Psychologically speaking, excitation of the premotor area together with area 8 would mean that an impulse is felt to reach for something or to look at it or to make some other movement. When these areas are damaged, we would expect that man or animal could no longer feel an impulse to move the affected limb. Several reports show that this is actually the case. After one "frontal eye field" was removed in a monkey, the animal showed what was called a "visual agnosia" in one half of both visual fields. When a banana was brought into the affected half of the visual field, the monkey seemed to see the banana but made no move to reach for it, though he avidly took and ate it as soon as it was brought into the normal half of his visual field (Fulton, 1951a). Another monkey, whose left frontal eye field was destroyed, did not take a banana he saw on the right side but took a peanut instead which was held out to him on the left side. (Monkeys always prefer bananas to peanuts, as did this monkey before the operation.) However, banana and peanut had to be shown at the same time, one on each side of the animal, before this disability could be observed (Pribram, 1955). In every case, the difficulty was temporary, probably because area 6 was preserved. We believe that this was not a visual agnosia but rather a lack of any impulse to move on the right side. This seems confirmed by another report (Stenvers, 1953). A patient with a tumor in the right frontal lobe which destroyed the motor cortex (hence produced a paralysis in his left arm) and also destroyed area 8, could not take an object shown within his left visual field though his right arm was not paralyzed. When walking in the woods, he was unable to keep from walking into trees on his left side, even though he saw the trees clearly. Here as in the case of Pribram's monkey, no impulse to move either toward or away from anything on the left side was felt, and thus obstacles on that side could not be avoided.

Appraisal of the action impulse. Unless an impulse to action is evaluated as appropriate in some sense and is liked, it would have as little chance of gaining attention and leading to action as a sensation that leaves us completely indifferent. If our earlier notion of a system that mediates the appraisal of sensations and action impulses is correct, we would expect the action circuit to connect with this system in its journey from hippocampus to motor cortex. More than that, we would expect that it is the fully organized pattern of action impulses that is appraised as good. Consequently, the connection with the system mediating appraisal (what we call the "estimative system") should be provided after the relays from the hippocampus have been organized in the cerebellum.

This actually seems to be the case. Cohen and associates (1958) have shown that the projection from the interposited and dentate nuclei of the cerebellum to the ventral thalamus (connecting with the frontal lobe) also goes to the centrum medianum, one of the intralaminar nuclei; and the projection from the fastigial cerebellar nuclei is relayed not only to the ventral thalamic nuclei connecting with the frontal lobe, but also to the midline and intralaminar nuclei, which are part of the estimative system.

The former pathway crosses over to the opposite side and makes discrete movements of arm, hand, or leg possible. The latter projection goes to both sides, mediating activity of the whole body. We have suggested that this projection connects with the extrapyramidal motor pathway which also serves emotional expression and action.

Apparently the primitive liking or interest in any movement whatever is mediated by the thalamic nuclei of the estimative system, which is responsible for the attractiveness of desire, just as it is responsible for the pleasantness of sensations. But to estimate whether this particular desire, the impulse to this particular action, is suitable or appropriate in the light of memories of similar actions, seems to require the cortical region of the estimative system, the anterior cingulate gyrus. This area connects with the hippocampus and so allows the recall of relevant visual, auditory, and other sensory memories necessary for correct evaluation.

Thus the estimative system seems to mediate a distinct quality of experience. It seems organized analogously to the sensory and motor systems in that the experience it mediates (liking and disliking) is consciously felt as soon as impulses reach the thalamus. But a correct appraisal on the basis of memory is possible only when impulses in this system reach the cortical receiving stations, the limbic areas. Unlike the sensory and motor systems, the estimative system does not provide accurate localization. By virtue of its connections with the sensory system we are able to appraise a particular object or experience as beneficial or harmful, so that we like or dislike it. Its connections with the somesthetic system in particular make it possible to realize that a given part of our body is giving us pleasure or pain, but this localization is not always precise. Electrical stimulation of the anterior cingulate gyrus seems to induce an appraisal that a movement of trunk or legs is suitable or unsuitable (by virtue of the connections from the motor areas for trunk and legs); stimulation of the posterior cingulate gyrus, an appraisal that sensations from these bodily regions are pleasant or unpleasant (by virtue of the connections from the sensory regions for trunk and legs). The movements actually made, intended to continue or change the appraised action and to prolong or avoid the appraised sensations, will include the parts of the body that are involved in action or sensation but may not necessarily remain the same whenever the same points in the limbic cortex are stimulated.

Direction of movement. To move toward or away from something requires an impulse to move, but also an impulse to move in a particular direction. When things are perceived to left or right, above or below, an action can be planned and the body and limbs organized to move in that direction. We have suggested that impulses indicating the position of the object are relayed via the corticopontinecerebellar tracts. But a continually changing location of the moving object requires a continuing relay of information while action is in progress. When a cat stalks a mouse, the impulse to action must be felt before action is taken and the stance adjusted in readiness for the pounce. But as the mouse moves, the direction of the pounce to come must change accordingly. Since the direction of action seems to be given by the direction in which the object is perceived and this information can also be relayed via long association fibers from sensory to motor areas, the direction of reaching or moving can be changed continuously.

That the spatial organization in various sensory areas does serve to indicate the direction in which to move or reach is shown by the fact that stimulation of a given point in the somatosensory area will arouse sensation in a particular spot on the periphery. Since the same spatial organization can be observed in the thalamus as well, it is understandable that some localization is possible even after ablation of the cortex. When the tail of a decorticated cat is pinched, for instance, the cat will bite its tail. To bite the pinching hand, the cat would have to be able to remember that it was the hand that pinched the tail—and that is impossible without the cortex.

The motor cells in the precentral cortex mediate a *set final position* of foot, ankle, knee, hip, etc. rather than contractions of specific muscles (Ward, 1952). Something seen above and to the left will be reached by activating a synchronized muscle pattern resulting in movement to the upper left. We have suggested that such synchronized patterns are relayed from the cerebellum; and that they are organized in the cerebellum on the basis of an appraisal for action, mediated by hippocampal activity. Pointing to a spot on the body surface that has been touched requires the whole action circuit from sensation to appraisal for action, organization of movement, felt impulse to move in a particular direction, appraising this impulse for suitability and direction, and finally executing the action.

A human being can correct the direction of movement when the movement has not brought the desired object within reach. Thus Stratton, in his famous experiment with reversed lenses, showed that he was able within a short time to reverse his movements according to the (now reversed) visual field. This experiment does not seem to have been tried with mammals, but a comparable experiment with newts has shown that reptiles are unable to reverse their native orientation. When the eyes of a newt were rotated in their sockets 180° without interrupting the optic nerve, the newt then struck upward instead of downward and so missed its prey every time. This reaction never varied during the lifetime of the animal (Weiss, 1952).

We suggest that the limbs are set in motion according to information as to the position of the object which is received in the cerebellum via relays from cortical areas. The direction of movement can be gauged and the movement adjusted continuously, as relays from cortical sensory areas arrive in the premotor and motor cortex via cortico-cortical association fibers. Indeed, the cortical sensory areas seem to serve the integration, localization, and fine discrimination of sensory impressions rather than the experience of a given sense quality. The thalamus seems sufficient for the experience of sensation as such.

Our analysis now allows us to explain some phenomena which have been observed after experimental ablation or after stimulation of frontal areas. For instance, when all cortex in front of the motor area is destroyed, the animal shows *groping,* that is, he makes repeated movements toward something he wants to reach (Fulton, 1951b). This seems to occur because the action initiated from the hippocampus and organized in the cerebellum cannot be accurately directed, for the area mediating the impulse to move *in a precise direction* is no longer available. The relay from cerebellum to motor cortex can initiate the action but the movement can no longer reach its aim. When vision is excluded, the appraisal of direction is no longer possible, and groping ceases. A similar interference with the aim of movement occurs with cerebellar lesions.

After destruction of all cortex in front of the motor area, the animal also shows *perseveration.* When given a piece of sugar, a monkey will keep on crunching rhythmically even after the sugar is gone. Apparently, the monkey cannot recognize that chewing is no longer necessary, an appraisal which seems to be mediated by the anterior insula, on receiving relays from the premotor cortex.

Our conception implies that smooth and effective action depends on an intact connection from the cerebellum to the premotor and motor cortex. The closer the lesion to the cerebellum, the greater will be the impairment because relays activating both the pyramidal and the extrapyramidal system will be interrupted. For this reason, there is greater interference with movement after removal of the cerebellum than there is after ablation of the motor cortex (see Chambers and Sprague, 1955). Of course, even after the cerebellum is destroyed, there will be some action because the brain stem reticular formation provides relay stations for impulses from the hippocampus, destined for the frontal lobe. When areas 4 and 6 are destroyed on both sides, the cortical end stations of this relay are missing and the movement has to be carried out via the connections from cerebellum to brain stem and spinal cord. Mammals like the rat or cat can stand and walk after decortication while monkeys can do neither. In primates, the cerebellorubrospinal connections are meager as compared with cat or rat.

Autonomic accompaniment. The involuntary muscles also have their share in the general mobilization for action. The active skeletal muscles must be provided with additional oxygen by increased blood flow at a time when the inactive inner organs need less oxygen. Hence, there will be a vasodilatation in skeletal muscles and a constriction in smooth muscles. When strong emotion urges to action, the autonomic changes are even more pronounced. There may be tremor, flushing or pallor, increased or decreased blood pressure, depending on the emotion and the action pattern to which it urges. There will also be endocrine activation, reinforcing and completing the emotional pattern. We will discuss these endocrine changes in connection with the various different emotional action patterns (see Chs. 6 and 7).

It would seem likely that the circuit which mediates the impulse to action will also mediate the autonomic and endocrine changes that go with it. Certainly, blood pressure and respiratory changes have been obtained from the limbic areas which, according to our scheme, constitute the first links in the circuit: Wall and Davis (1951) have obtained blood pressure changes on stimulating the anterior cingulate gyrus, the insula, and also the sensorimotor and orbital cortex in anesthetized monkeys. In waking animals, autonomic changes have been observed together with emotion on stimulating the limbic system and the hippocampus (Kaada, 1951). Further along our proposed action circuit, in the brain stem reticular formation, the hypothalamus, and midbrain, electrical stimulation has also evoked blood pressure changes and other autonomic responses at widely scattered points. According to Rioch:

> The diffuse scattering of the descending pathways . . . is rather striking. It suggests that there are not separate, single vasopressor, vasodepressor, pupillary, pilomotor, sweat etc. centers in the hypothalamus, but instead that the type

> of localization in the hypothalamus, subthalamus and midbrain is of complex patterns of behavior, e.g., . . . rage, fear and fighting, female oestral behavior etc. Evidence from localized electrical stimulation in the mesencephalon and remaining diencephalon of decorticate cats support such a conclusion. (1940, p. 284)

If autonomic changes are mediated by neural relays that are incorporated into the various action patterns, it would seem likely that these relays run via both pyramidal and extrapyramidal routes. Various research findings admit such an interpretation.

For instance, it has been shown that stimulation of the motor cortex induces a psychogalvanic response (PGR) which is abolished by section of the cerebral peduncles (Wang and Lu, 1930), but not by destruction of the hypothalamus. This means that the motor cortex may receive relays from the hypothalamus initiating the PGR, rather than that the hypothalamus receives relays from the motor cortex and relays them to spinal centers. And Landau (1953) found that stimulation of the medullary pyramidal tract produced sweating, changes in blood pressure and pulse rate, contraction of pupils, nictitating membrane and bladder, and also piloerection. Hence it seems that the motor cortex and pyramidal tract relay impulses to autonomic motor nerves just as they do to somatic motor nerves.

However, the motor cortex and the pyramidal route, as we have seen, only mediate discrete movements of the limbs, while the extrapyramidal pathway from the premotor area to the globus pallidus and spinal motor neurons seems to mediate movements of the whole body, the background to discrete limb movements. Hence autonomic changes should also be mediated via both the pyramidal and extrapyramidal pathways. The corticobulbar tract, originating from the motor cortex, connects with the motor nuclei of the cranial nerves mediating parasympathetic activity, while both pyramidal and extrapyramidal pathways connect with the sympathetic nervous system through the spinal cord.

This suggestion is supported by the finding that the psychogalvanic response to a whistle, a pencil tap, or the like occurs even after the motor cortex is destroyed, provided that the premotor area is intact. In fact, this seems to be the only area absolutely necessary for the reflex; for the PGR persists as long as area 6 is intact, even though the rest of the cortex, including the motor cortex, is destroyed (Schwartz, 1937). This would suggest that the PGR is also mediated over an extrapyramidal pathway and accompanies any impulse to action, voluntary or involuntary, deliberate or emotional. A casual reaction to sensory stimulation, startle, and different emotions, all are accompanied by a psychogalvanic deflection; but this deflection is largest when there is a *sudden* impulse to action.

Not only the PGR but other autonomic changes also seem to be mediated via cortical areas, and probably are relayed over both pyramidal and extrapyramidal pathways. For instance, gastric peristaltic movements occur on stimulation of area 8 (Davey *et al.*, 1949) and inhibition of peristalsis on stimulation of area 6 (in recently fed monkeys; Sheehan, 1934). We would suggest that these movements occur when food is perceived, focused on, and wanted. Salivation occurs under the same circumstances and can also be induced by stimulation of area 8. An inhibition of peristalsis ordinarily occurs when there is a strong impulse to action, which demands muscular activity and so a reorganization of the bodily economy. The impulse to action is duplicated by stimulation of area 6.

Stimulation of the orbital surface of the brain induces respiratory arrest and changes in stomach motility (Babkin and Speakman, 1950; Kaada *et al.*, 1949) which seem to be the autonomic changes that go with eating. These changes also occur when something is smelled, appraised as good to eat, and is about to be eaten. Stimulation of the orbital cortex, the olfactory association area, could produce these changes.

Vasodilatation and constriction. From a great deal of evidence in this area it is possible to infer that dilatation or constriction of blood vessels is brought about when a pathway is excited which runs from the midbrain via the hypothalamus to the frontal lobe. It is generally agreed that both dilatation and constriction of blood vessels is produced via sympathetic fibers. When these fibers are excited, the blood vessels constrict, the more intensely, the stronger the excitation. When these fibers are not excited, the blood vessels relax. But the blood vessels of skeletal muscles seem to be innervated by special sympathetic vasodilator fibers that are cholinergic in function and actively dilate the vessels; the diameter of such dilated vessels is much greater than mere relaxation could accomplish. These sympathetic vasodilator fibers are excited whenever striped muscles are innervated, that is, whenever there is any movement (Lindgren and Uvnäs, 1955; Folkow, 1955).

Eliasson and his coworkers have found that there are two regions in the medulla oblongata which induce such dilatation of blood vessels. The area in the ventrolateral part of the medulla induces active (bilateral) dilatation of blood vessels in the muscles,* and is connected with the supraoptic region in the hypothalamus and with the motor cortex (Lindgren and Uvnäs, 1955; Eliasson *et al.*, 1952, 1954). A cut several millimeters deep behind the optic chiasm which extended several millimeters beyond the midline on both sides did not eliminate these changes when the supraoptic area was stimulated. This seems to indicate that the pathway relays in the direction from the midbrain to the hypothalamus and motor cortex rather than in the reverse direction as the authors assume; for it was interrupted behind the point of stimulation, yet the lesion did not abolish the response. It seems likely that vasodilator impulses together with motor impulses are relayed in the action circuit from the hippocampus to the cerebellum, hypothalamus, and frontal lobe, and that they finally connect with the motor cortex and pyramidal tract (cf. McQueen *et al.*, 1954). This is confirmed by Wall and Davis (1951) who report that vascular responses to stimulation of motor and sensory cortex are unchanged after destruction of the hypothalamus but disappear when the pyramids are sectioned. When the animal is decerebrated by a cut through the superior colliculi, electrical stimulation of the midbrain, which before the operation had induced a dilatation of the blood vessels in skeletal muscles, did so no longer. This operation had interrupted the connection between midbrain and motor cortex.

However, ablation of the motor cortex does not abolish the vasodilatation produced by stimulation of the preoptic area in the hypothalamus (Eliasson *et al.*, 1954), which seems to suggest that this area is connected with the extrapyramidal pathway also. The extrapyramidal route seems to be activated too by stimulation of the anterior temporal lobe which, as we know, excites the amygdala and hippocampus. The blood pressure changes that occur on such stimulation would probably accompany emotional reactions in the unanesthetized animal. In the animal under narcosis, the blood pressure responses that occur on stimulation of the anterior temporal lobe seem to be independent of both hypothalamus and pyramidal tract (Wall and Davis, 1951) and seem to employ a direct extrapyramidal route.† Normally, intense excitation of the anterior temporal lobe and amygdala

* But *contraction* of vessels in skin and internal organs, and hence a *rise* in blood pressure.

† See Fig. 6, p. 72, and Fig. 7, p. 83.

is experienced as hallucination or perceptual distortion and may excite intense emotions (cf. Heath *et al.,* 1954); hence, large blood pressure changes would be expected via the route from amygdala to brain stem.

When the uncus, temporal cortex, and amygdala were removed on one side in animals, stimulation of the cingulate gyrus on the opposite side reduced the blood pressure changes by 90 percent; stimulation of the cingulate gyrus of the same side reduced them by 10 percent. Hence, Wall and Davis suggested that the pathway mediating these changes runs from the cingulate gyrus via the anterior temporal cortex to the sympathetic effectors. We would assume that the route is rather from the cingulate gyrus via the cingulum to the hippocampus, brain stem, and frontal lobe; when activated it arouses an emotional action tendency (startle or fear). When the uncus, temporal cortex, and amygdala are removed, a sound can no longer be identified and will not induce fear. Even after removal of the amygdala, stimulation of the cingulate gyrus, which—according to our scheme—mediates the appraisal of action, may induce the animal to stop moving or to change his action, but now the new activity can no longer be guided by imagination, and so emotion will be mild and action feeble. Removing the anterior temporal lobe with the amygdala seems to exclude imagination and the action impulses and autonomic changes induced by imagination.

Of course, autonomic reflexes can occur independently of emotion, hence independently of the action circuit we have described. The blood pressure and respiratory mechanisms that function reflexly apparently need only an intact connection between afferent and efferent fibers in the medulla oblongata. But when these functions are influenced by emotion and action, this influence seems to be exerted via the action circuit. The pyramidal connection seems to mediate the autonomic changes necessary for discrete movements while the extrapyramidal connection seems to mediate the autonomic changes of the body as a whole, as required for emotional expression and action.

4. REGISTRATION AND INHIBITION OF ACTION

We have tried to sketch the sequence from perception to emotion and action and have suggested that action is mediated by a relay from sensory cortical areas via subcortical structures to the cortical motor areas. This circuit seems to mediate action and also to serve the registration of action.

REGISTRATION OF THE MOTOR PATTERN

Every action is remembered, that is, registered and retained. This means that movements must be registered in sequence and retained in structure. Just as sensory impressions are registered in the sensory association areas whenever sensory impulses are received in the primary sensory area, so movements will have to be registered in the frontal association areas whenever an impulse to move is felt or an actual movement is made. We have argued that we feel an impulse to action when neural relays from the hippocampus and cerebellum arrive in the premotor area, and that a movement is carried out when the combined impulses from the cerebellum and premotor area are strong enough to activate motor cells.

If there is any similarity in organization between the motor and sensory projections, we would expect that the motor projection from the cerebellum to the frontal lobe would mediate not only the experience of impulses to action but would also serve to register such impulses and to allow their appraisal and its registration. Even when a movement is only imagined, it seems to be registered in the prefrontal area and to activate the premotor and motor cortex sufficiently to bring about action currents in the muscles. Jacobson (1932, 1934) has shown that such action currents can be recorded from the arm or leg muscles when a man imagines moving arm or leg, and from the eye muscles when he imagines looking at something. There is also evidence that a skilled act can be learned by imagining it in detail. Thus Beattie found that dart throwing could be learned just as effectively by imaginary practice as by real practice (see Arnold, 1946). This would indicate that imagined movements must be registered like actual movements.

We have seen that the projection from the cerebellum to the frontal lobe is relayed via the brachium conjunctivum and the ventral thalamic nuclei (Ch. 3). While the lateral ventral nuclei connect with the motor and premotor cortex, their anterior and medial parts connect with the prefrontal areas (Cohen *et al.*, 1958; Whiteside and Snider, 1953), probably via the caudate nucleus (Papez, 1956; Showers, 1958). It would seem that the lateral ventral nuclei mediate the felt impulse to action and also activate the motor cortex, while the medial and anterior portions of these nuclei serve the registration of the pattern and sequence of movements (whether imagined or carried out) in the appropriate areas for face, hands, arms, trunk, or legs (see Figs. 5, 6). At the same time, the projection to the thalamic estimative system seems to mediate the appraisal of action impulses and the registration of their success or failure (see p. 94 f.). Damage of the medial thalamus has impaired a single alternation habit in monkeys (Rosvold *et al.*, 1958), apparently because the animals could no longer appraise their action impulse as suitable or unsuitable and so did not know what to do.

Motor recall. To reproduce a complicated action faithfully, it must be well practiced until it is almost automatic. This holds for the reproduction of limb and trunk movements as much as for the reproduction of speech. With successive repetitions, the sequence and structure of the movement is gradually perfected and so retained. Until the sequence is perfect in every detail, we use imagination whenever memory fails. This accounts for the "reconstruction" of memories investigated by Bartlett (1932). It is only as we become proficient that we can forego the attempt to imagine what to do next and come to depend on the automatic reproduction of the correct sequence. Once learned, an action pattern seems to be activated as soon as the cue is given, without resorting to imagination. Indeed, thinking about it often disturbs recall, and so does any distraction that disturbs the motor sequence. A singer may recall a song perfectly if he sings it but may fail miserably if he is asked to say the words. A well-learned speech or poem may be reproduced without difficulty, though a man may be unable to recite the second paragraph or stanza without the first.

Motor imagination. Motor imagination, like sensory imagination, is a recombining of memory images to serve the purposes of the moment. But visual and auditory images lend themselves far more aptly to a creative rearrangement of colors, scenes, or tones. The visual arts and music depend on such creative efforts which are limited only by the type of memory images a man may have at his disposal. A man born blind cannot imagine color, a color-blind person cannot imagine what the world looks like to his normal fellows, and someone born deaf has no conception of melody. Even when there is no such sensory defect, some people's visual or auditory memory is poorly developed; their visual and auditory imagination will be similarly restricted. Motor imagination also allows creative application, as in working out a dance pattern or writing a poem. In a more mundane way, everyone uses motor imagination in learning motor skills. Abstract and sense knowledge must be translated into motor images before a new movement can be carried out correctly. The golf pro may tell the novice how to hold a club and how to swing, but the learner must plan his action and imagine the motion if he is to do it correctly. If he does not, he lets himself be guided by old and now useless motor patterns instead of learning a new and appropriate motion.

The activation of motor memory. Motor memory, like sense memory, seems to be activated via the hippocampal circuit. But the motor memory circuit seems to include the cerebellum and its projection to the ventral thalamic nuclei, for the reproduction of a movement involves the same pattern of limb and trunk muscles as the original movement. The neural impulses relayed from the anterior and medial ventral thalamic nuclei to the prefrontal cortex seem to be passed on to the premotor and motor cortex, and so facilitate and guide the movements to be made. According to Krieg (1954), the number of cortico-cortical association fibers conducting from the prefrontal areas to the motor area increases progressively from the frontal pole to the precentral gyrus. Apparently neural impulses arriving in the prefrontal area are conducted toward the motor cortex, which may explain why there is no degeneration of the prefrontal cortex after prefrontal lobotomy. It may also account for the fact that motor habits are not disturbed by such an operation. Apparently, the intracortical conduction from the prefrontal to the motor cortex is sufficient to produce the facilitation of overlearned actions. We would expect, however, that a new motor habit might be difficult to learn if it required movements registered in areas isolated by the lobotomy.

Earlier psychologists often assumed that the facilitation of action could be explained as a modification of peripheral synapses. Against this notion speaks the fact that the same synapses are activated for very different movements. Playing the piano and typewriting seem to exercise similar muscles, but a good typist is not necessarily a good pianist. Still less can the performance of a concert pianist, who has a prodigious repertoire, be explained by peripheral facilitation. But it can be explained as the reactivation of a pattern laid down in the prefrontal cortex. Its registration ensures an accurate temporal and spatial sequence which can eventually be reactivated at will. The function of practice seems to be not only the facilitation of each movement but also, and more importantly, the activation of the correct pattern via the hippocampus. Our explanation would account for the fact that the prefrontal cortex is so extensive in man, even compared with the chimpanzee. The human being has a far greater repertoire of actions than any other mammal, including the monkey and ape.

Some recent animal experiments offer support for our hypothesis. Rosvold and associates (1958), for instance, found that bilateral lesions in the caudate nucleus, the medial thalamus, and the splenium of the corpus callosum seriously impaired single alternation habits in monkeys. The lesions in the medial thalamus and the splenium also damaged the hippocampal commissure or fornix and thus interrupted the memory circuit. Single alternation problems force the animal to recall what he did before, and the lesions made such recall impossible.

Stimulation of the caudate nucleus either during, before, or after a trial impaired the acquisition of a reversal habit in which cats had to learn that the object to the right was baited and, as soon as learned, that now the object to the left was baited (Thompson, 1958). This required recalling that going right would be successful at first, going left, later. Electrical stimulation of the caudate nucleus apparently disturbed the activation of memory traces in the prefrontal cortex.

The activation of motor imagination. Motor imagination, like sensory imagination, implies a manipulation of memory images, even though this may not be extensive. Motor imagination is restricted to a new adaptation of fundamental patterns of motor coordination, and does not allow entirely new combinations, as is the case in the visual and auditory sphere. Motor images, like sensory images, seem to be activated via the amygdalar circuit. The amygdala is connected with the dorsomedial thalamic nucleus which sends relays to the frontal cortex. According to Meyer *et al.* (1947), the small-celled larger portion of the dorsomedial nucleus projects to the lateral part of the orbital region (area 47 and lateral 11) and to the frontal gyri, namely, the inferior gyrus (areas 44, 45, 46), middle (areas 46, 9, 10), and superior gyrus (areas 8 and 9).*

We have seen before (Ch. 3) that the imagination circuit is needed to learn a complicated motor habit. Bilateral destruction of the amygdala or of the dorsomedial thalamic nuclei make it impossible for animals to learn a complicated sequence of actions. But a simple motor habit is not lost after interruption of this circuit or destruction of various sensory association areas. Of course, destruction of the prefrontal cortex abolishes motor imagination together with motor memory.

Every emotion is an urge to action, either a tendency to continue the present state or to change it. Fear, for instance, is an impulse to run away or withdraw, and is accompanied by various bodily changes. Both the impulse to escape and the simultaneous autonomic motor impulses seem to be registered in the prefrontal areas, to be reactivated whenever a similar experience arouses a similar fear. We may not even be aware that this situation is similar to something we have experienced in the past. As long as the appraisal for action is similar, the retained motor pattern, both skeletal and autonomic, will be reactivated and will reinforce the emotion and intensify its physical symptoms.

When we recognize something that will bring pain, for instance, the dentist's drill, we are afraid. This fear is not merely the immediate reaction to danger, it is a reliving of many old fears. We feel the old tension, the old malaise. This reactivated action tendency, unless counteracted by other experiences, grows stronger with every repetition and may develop into an enduring attitude. There is little advantage in calling this attitude "unconscious," for it is not continuously active underground, nor is it repressed. As long as the motor engrams of earlier emotions are not reactivated, they are dormant. However, the appraisal of something as good or bad also leaves a residue, a memory trace in the limbic cortex, which disposes to a similar appraisal and a similar emotion. Accordingly, memory traces of what has been liked or disliked, and of what has been done in the past, influence both appraisal and action impulse.

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* The large-celled (but smaller) part of the dorsomedial nucleus receives fibers from the nuclei of the midline and the median center nucleus of the thalamus, and has a two-way connection with the hypothalamus via the periventricular system (Kuhlenbeck, 1954; Rand, 1954). According to Meyer and associates (1947), this extreme medial part of the dorsomedial nucleus sends fibers to the medial half of the orbital region (areas 12 and medial 11). We would suggest that this projection serves olfactory imagination.

PREFRONTAL LOBOTOMY

It is now possible to discuss the effects of an interruption of fibers from the thalamus and hypothalamus to the prefrontal cortex, such as occurs in prefrontal lobotomy. According to Freeman and Watts (1948) and Meyer and Beck (1954), standard lobotomy is followed by complete degeneration of the dorsomedial thalamic nuclei and by partial degeneration of the anterior and ventral thalamic nuclei, depending on the extent of the cut.

Reduction of the impulse to action. Prefrontal lobotomy produces a significant loss of alertness, a general slowing and inertia (Kalinowsky *et al.*, 1952). This is not noticeable in simple actions like finger tapping, but the speed of decision is strikingly impaired (Landis and Clausen, 1955). In fact, Landis and associates (1950) have suggested that lobotomy produces not only the well-known decrease in alertness and anxiety but also a decrease in enthusiasm and general interest. The patient will often sit quietly without doing anything at all for long periods of time. This deficit is most striking after a posterior cut which isolates a considerable part of the premotor cortex. We would explain it as a pronounced reduction in the number of action impulses that are experienced.

Deficit in motor memory and motor imagination. There is a significant deficit in the Porteus maze test, the Bender visual gestalt test, and the Koh's block test (Freudenberg and Robertson, 1949; Kalinowsky *et al.*, 1952; Porteus, 1959). A decided loss in creative ability has been reported also (Freeman and Watts, 1942; Ashby and Bassett, 1949). These defects can be explained by the interruption of the motor memory and motor imagination circuits. In addition, memory registration also has been curtailed. After lobotomy, action impulses can neither be felt to the same extent, nor can they be registered or reactivated. Consequently we would expect that movements can no longer be either recalled or imagined. Though this defect is not nearly as incapacitating in man as in animals, because the human being can think abstractly and remember concepts and visual directions according to which he can move, yet the lack of motor imagination is bound to influence motor performance. Hence patients often complain that they have lost their facility in drawing or painting (Dax and Smith, 1948). They are said to have no "foresight" because they cannot imagine their actions before they act. They cannot project possible actions in imagination and so cannot initiate plans for action.

Goldstein (1944, 1949) suggested that prefrontal lobotomy, like frontal damage, produces a loss of the "abstract attitude." This concept grew out of his observation that patients with frontal lobe injuries apparently could handle concrete situations better than abstract concepts. For instance, a patient might be able to drink a glass of water or comb his hair, yet not be able to pretend drinking water or combing his hair. But surely this defect is not an inability to comprehend the concept of "drinking" or "combing." It can best be understood as an inability to imagine the appropriate action. Goldstein also found that lobotomized patients were unable to select objects that belong together according to more than one category, while others had difficulty grouping items together that belong together.

We would think that the deficit actually found was not a loss of abstraction but an inability to employ motor imagination. The instructions of the test seem to decide whether the performance will be normal or impaired. If the patient is given pieces of colored wool and told to "pick out all the yellows and put them here" (pointing to a spot on the table), he may have no trouble. But if he is asked to "sort them" or "order them" according to color, he may have insuperable difficulties, not because he does not understand the abstract concept but because he cannot project the action in imagination and so does not know how to start and what to do next. This difference in instruction accounts for the negative results obtained by King in the Columbia-Greystone studies (Mettler, ed. 1949).

Reduction of anxiety. In spite of such general slowing down of action, prefrontal lobotomy has been used with psychiatric patients because it reduces and sometimes eliminates anxiety. After lobotomy, the patient reports that he does feel pain or fear or other emotions but is no longer bothered by them. According to our interpretation, he is now able to deal with these emotions because an emotional urge *as it occurs on any given occasion* is not intense enough to overwhelm him, as long as the emotion reinstated from earlier experiences is not added to it. Now the patient sees the dentist's drill, remembers the pain it has caused in the past and knows that the drill will hurt again. He feels the urge to get away, but this urge is no longer reinforced by the living memory of former impulses to escape and by reinstated autonomic changes.

According to Meyer and Beck (1954), the projection from the dorsomedial thalamic nucleus enters the frontal lobes in the ventromedial quadrant; hence the excellent results of lobotomies confined to both these quadrants. This as well as the standard cut will prevent the reinforcement of action impulses by memory and thus free the patient immediately from the reinstated urge to escape, that is, from anxiety. After operation, every danger will be judged on its own merits and fear will lose the compulsive force that comes from habit. When there is no known source of danger, there will now be no fear, no impulse to escape. But if there should be a persisting cause for anxiety or a persisting habit of appraising new situations as dangerous, the patient may still experience constant fear even though the operation has relieved him of the cumulative effect of earlier fears.

As long as there is any connection left between the ventral thalamic nuclei and the prefrontal region, we believe that it is possible to form new memory traces, even though the number of fibers projecting to the prefrontal cortex may be reduced after lobotomy. In addition, the relays from the prefrontal cortex to the motor region are preserved, which may account for some facilitation of action. Thus anxiety may return some weeks or months after lobotomy. If it does, a more posterior cut or the bilateral destruction of the dorsomedial and anterior thalamic nuclei may bring relief (Spiegel and Freed, 1949). According to our hypothesis, lesions of the dorsomedial nucleus would prevent motor imagination and hence also images of wanting to escape; lesions of the anterior nucleus would prevent the imaginative appraisal of successful and unsuccessful actions, and hence also the worry about what should be done and what is better left undone. Ablation of the medial and anterior portion of the ventral thalamic nuclei should prevent reactivation of motor memories, which would most effectively reduce anxiety.

Prefrontal lobotomy seems to prevent not only the reinstatement of action impulses but also the reactivation of the autonomic changes that go with emotion. Elithorn *et al.* (1954) found, for instance, that twelve out of thirteen patients had a significantly smaller psychogalvanic reflex (PGR) to a light announcing an electric shock, though the shock itself produced a response as large as it had been before the operation. This seems to confirm that the cumulative effect of action impulses is abolished after lobotomy, though the emotional urge to action and the autonomic changes that go with it can be aroused afresh. The continual reinforcement of autonomic impulses by a reactivation of autonomic motor engrams also seems responsible for the vasoconstriction and high blood pressure found in anxiety. Lobotomy, which interrupts the projection from the ventral thalamic nuclei to the prefrontal cortex, prevents such reaction. Hence it brings about a dilatation of blood vessels in the skin so that the limbs become pink and warm (Freeman and Watts, 1942).

Orbital undercutting produces hyperactivity. The animal paces restlessly in his cage and drops off to sleep suddenly, without the usual preliminary period of rest. Wall *et al.* (1951) have shown that this is the result of cutting an unmyelinated tract that runs almost perpendicularly from the orbital surface to the caudate nucleus. We shall see later in this chapter that this tract seems to mediate relaxation; interrupting it would result in restless activity. Even after undercutting the orbital region, electrical stimulation of the orbital cortex has induced vasomotor changes. Apparently, vasomotor impulses can be relayed by intracortical fibers from the orbital cortex to the septal region (cf. Krieg, 1954) and connect via the amygdala with effector pathways.

The *emotional flattening,* which is a conspicuous result of lobotomy, can also be explained as the inability to revive earlier emotions. Not only fear is cumulative, eventually forming a backlog of anxiety, but active interest in things and people also depends on a background of reinstated action impulses. An emotional attachment is alive and profound only as long as a man feels a continuing desire to be with his friends, to do and say things that please them. The sentiment of love, like the abiding enthusiasm for a cause, requires a reliving of the emotion whenever we encounter anything connected with our love or interest. The lobotomy patient may still like to be with others and enjoy their company. Given some particular need, he may even make an active effort to see his friends, but he has lost the steady outgoing interest that bespeaks a profound attachment.

Interference with the appraisal of action. The reported "loss of inhibition" after prefrontal lobotomy seems to result from the interruption of fibers to the anterior cingulate gyrus. The patient will give in to every impulse as soon as it arises and persist in the same inappropriate action. This accounts for the carelessness and tactlessness of lobotomy patients.

Yakovlev and his coworkers (1950) have found that the cingulate gyrus, the orbital limbic area, and the insula are involved in prefrontal lobotomy, which accounts for inappropriate speech as well as inappropriate actions. Such damage probably accounts also for the urinary incontinence frequently found shortly after lobotomy (Greenblatt *et al.,* 1950). The urge to urinate is not immediately recognized as inappropriate and so is given in to, until the patient learns to act on the first sensation of a full bladder.

The injury to the cingulum also seems to account for the fact that lobotomized patients seem to be much more sensitive to heat, pricks, or cuts, though intractable organic pain seems to be reduced (Chapman *et al.,* 1948; Malmo, 1948). There is no doubt that the patient feels the immediate pain, whether organic or inflicted from the outside, and feels a strong impulse to escape from it. Organic pain seems to be less bothersome because there is no longer a cumulative effect and the patient takes each pain as it comes without anticipating it in imagination or being overwhelmed by the reactivation of earlier impulses to escape. When a hypodermic is given or his reaction to heat is tested, there is an immediate impulse to withdraw from the pain. The normal person can immediately appraise this impulse as inappropriate, for he knows that the hypodermic is necessary and the heat test useful, and thus he inhibits flinching. The lobotomized patient is unable to make such an intuitive estimate and impulsively withdraws from heat or pain.

CINGULECTOMY AND CINGULOTOMY

In this operation, the defect is the result of interrupting the projection from the anterior thalamic nucleus to the cingulate gyrus. Hence, we would expect the patient to be unable to appraise his actions intuitively. However, since the anterior insula is not affected, the intuitive appraisal of speech remains, accounting for the report that this operation does not result in loss of inhibition. Both the undercutting and the destruction of the anterior cingulate gyrus seem to have reduced the agitation of obsessional neurotics, though they do not influence anxiety states (LeBeau, 1952). The obsessional neurotic is agitated because his actions seem to him unsatisfactory, imperfect, so that he repeats them over and over. After the operation, he can no longer automatically appraise his action and find fault with it, and so he will no longer try to improve it by repeating it. For the same reason, obsessive and ritualistic behavior disappears, but there is also less tolerance for petty annoyances and irritations (Tow and Whitty, 1953). We would expect, however, that obsessive speech mannerisms would not be improved by cingulotomy or cingulectomy.

There are some reports that destruction of both cingulate gyri has resulted in "akinetic mutism" (Nielsen, 1951; Barris and Schuman, 1953). The patients were at first confused, excited, and aggressive, but gradually became completely apathetic, would not talk, eat, or move, became stuporous and finally died in coma. Since therapeutic ablation of the cingulate gyri does not usually have such untoward effects, it seems likely that a secondary degeneration of the midline and intralaminar nuclei was responsible for this development.

Experimental results of animal studies also confirm our hypothesis. Stamm (1955), for instance, reported that lesions of the medial cortex in rats (which includes the homologue of the anterior and posterior cingulate gyrus and the retrosplenial region in man) resulted in striking behavior changes. These rats did not let their young suckle, seemingly because such tactual sensations were no longer felt as pleasant. The mother rats no longer deposited their young in one spot away from airblast, or covered them up with paper strips to protect them from heat, as did normal rats. These rats ran around aimlessly when exposed to airblast or heat, picked up their litter, but soon dropped the young again in any part of the cage. Apparently, they were still able to estimate that heat or airblast is harmful, and knew enough to pick up their young (the lesions only amounted to 16 percent of the total medial limbic cortex, and the homologue of the insula was intact). But their estimate as to where to run was completely disorganized, which accounts for the aimlessness of their actions.

This analysis of the effects of psychosurgery may help us to understand the puzzling "personality changes" that appear after some brain lesions, and may also help to throw light on the neural substrate of attitudes and sentiments. Without the possibility of reinstating earlier motor memories, a man's emotions find no reverberation and are cut off from anything that has moved him in the past. He may still have likes or dislikes (mediated by the estimative system), but he is no longer able to translate them into actions that require planning or imagination. At the same time, he can act routinely or on the basis of a momentary impulse. If he has established desirable habits of action, the "personality change" may be minimal. If he has not, he must act planlessly, on the basis of a momentary impulse, which results in the well-known "disinhibition." This explains the finding that the outcome of lobotomy depends on a patient's prepsychotic personality. But even in the best of cases, emotions will be shallow and fleeting.

After cingulectomy or cingulotomy, the outcome will depend on well-established habits of appraisal and action. Though the intuitive appraisal of action is no longer aided by the memory of earlier successes and failures, the patient can still appraise the situation and plan his action. If he is used to planning his action and to acting according to definite principles, he will appear normal.

THE INHIBITION OF ACTION

Thus far, we have described the circuits that serve the initiation of action. Once an action is begun, a new appraisal is required if that action is to be stopped. Even before the action, at the point when the impulse to it is felt, this impulse must be appraised as appropriate or inappropriate so that it can either be carried out or inhibited. When an action impulse or an action is to be inhibited, the appraisal leading to such inhibition will have to initiate neural impulses that eventually reach the motor cortex, motor nerves, and muscles, not to excite but to inhibit them.

Surprise as inhibition of action. Psychologically speaking, there are several ways in which action can be inhibited. There is, for instance, the arrest of action that comes from surprise. A sudden encounter may present us with the demand for an entirely different action. Until we can decide on the best thing to do, all movement is held in abeyance so that the situation can be appraised afresh.

We have suggested before that the appraisal of situations as well as actions is mediated by the estimative system which receives relays from the sensory and motor systems (see Ch. 3). When the circuits serving the intuitive appraisal are preempted by electrical stimulation, it is impossible to appraise what is perceived and hence impossible to act. The result would be a state of mind much like that in surprise before the unexpected thing can be appraised. This seems to explain the arrest of action obtained in waking animals by fast frequency stimulation of the intralaminar nuclei in the medial thalamus (Hunter and Jasper, 1949).

Success as completion of action. When the goal is achieved, the action is completed. Thus man or animal stops eating when the food is gone or his hunger stilled. Such an arrest of action presupposes an appraisal that the ongoing action is no longer suitable and must be arrested (via connections from the anterior cingulate gyrus to hippocampus, cerebellum, and frontal lobe).

Since the impulse to action seems to be experienced when neural impulses arrive in the premotor area, and the appraisal of action occurs when impulses are relayed to the anterior cingulate gyrus, this area should play a special role. Indeed, stimulation of the anterior cingulate gyrus (area 24) produces immediate arrest of movement, as does also stimulation of the motor area connected with it. Thus stimulation of areas 4s and 8s (the so-called "suppressor strips" in front of the motor area and of area 8) has resulted in the immediate suppression of movement (Penfield and Jasper, 1954). We have suggested that the premotor area, comprising areas 8 and 6, mediates the felt desire to move, which is appraised as suitable or unsuitable when neural impulses travel from there to the anterior cingulate gyrus. Area 4s, just in front of the motor area, seems to relay neural impulses to the anterior cingulate gyrus which allow the appraisal of ongoing movements; while area 8s, just in front of area 8, seems to send relays which allow the appraisal of action impulses. It has been reported that stimulation of these two suppressor strips and of the anterior cingulate gyrus relaxes existing muscular contractions and interrupts electrical afterdischarges so that movements seem to melt away (McCulloch, 1944; Bailey *et al.*, 1944).

McCulloch has reported suppressor areas also in the somatosensory cortex (2s) and in the visual cortex (19s), though the induced suppression is usually not an arrest of action but merely a suppression of spontaneous electrical activity in the cortex. It has been objected that such suppression is produced when the cortex is exposed to air for a long time, as is the case in stimulation experiments (Meyers *et al.*, 1954), but McCulloch has shown that it must depend on neural conduction because it appears when impulses from the suppressor areas reach the caudate and ventrolateral thalamic nuclei. If so, the stimulation of these suppressor areas seems to simulate an appraisal that something seen, heard, or felt now demands a different action; this arrests the movement about to be made.

Meyers and his associates have also objected that the stimulation of suppressor areas brings about an arrest of action only in the anesthetized and not in the waking animal, and so cannot represent normal activity. But we have attempted to show that there has to be a mechanism that can arrest action, just as a mechanism is required which can initiate it.

Of course, anesthetics make the *experience* of the appraisal for action impossible, but this does not mean that the pathways mediating such an experience cannot be stimulated electrically when the animal is unconscious. In fact, we would expect pronounced effects during anesthesia because all spontaneous appraisals are excluded and the pathways serving appraisals are inactive. In the waking animal, the stimulation of suppressor areas in the neocortex excites pathways which have to compete with many others before action is decided upon, or before it can be arrested.

Rest and relaxation. Action is also arrested when emotion brings about muscular relaxation. Psychologically speaking, this relaxation follows the experience of "not wanting to move" when the emotion is pleasure, or of "not being able to move" when it is extreme fear or terror. The experience of weakness that accompanies fear seems to be the result of increased adrenaline secretion which depresses synaptic transmission (see Ch. 7). Hence, the paralysis of action in terror is not only the result of the physiological changes during emotion, but also the result of an appraisal that the danger is so great that effective action is impossible. This appraisal inhibits movement even before the autonomic effect of terror can be felt.

Muscular fatigue can induce an appraisal that further movement is undesirable, and man or animal will rest and relax. Such muscular relaxation may be the result of an intuitive, unwitting appraisal; but, at least in man, it may also be induced deliberately and achieved by training (cf. Jacobson's "General Relaxation," or Schultz's "Autogenic Training"), which may include smooth as well as striped muscles. In animals, complete relaxation is usually the prelude to sleep, and even in man sleep will follow unless awareness is maintained deliberately.

Both the decision "not to move" when movement has achieved its purpose or is found inappropriate and the experience of "not wanting to move" require neural impulses that will counteract motion. So does a decision to induce partial or general relaxation. Now we have suggested that all motor impulses are initiated by an appraisal for action which is mediated by the hippocampus. Thus we would expect that the decision to stop action or to rest and relax, no less than the decision to act, is mediated by this structure.

Moreover, we would postulate that the same circuit which mediates action would also mediate the arrest of action and the relaxation of muscles. Indeed, action itself requires not only the contraction of some muscles but also the relaxation of others. If action is to be smooth, there must be some provision for motor inhibition as well as for motor excitation. Since we conceive that action impulses are organized in the cerebellum, we would expect that this structure would coordinate inhibitory as well as excitatory impulses into one action pattern.

Inhibition in the cerebellum. It has been shown that high frequency stimulation (300 per second) of the cerebellar cortex results in facilitation of a movement produced by stimulating the cerebral motor cortex, while low frequency stimulation (10 per second) inhibits that movement.* The inhibitory effect is relayed via the dentate nuclei to the ventromedial reticular formation in the caudal part of the medulla oblongata, while the facilitatory effect is transmitted via the fastigial nuclei to the lateral reticular formation lying just in front of the inhibitory area (Nulsen and associates, 1948).† However, Moruzzi (1954) has shown that polarization of the anterior lobe of the cerebellum (which inhibits movement) *prevents* the firing of some medioventral bulboreticular cells but *augments* the activity of others, and *excites* some neurons that had been inactive. This would imply that a pattern is activated which includes individual units in both parts of the reticular formation. Since the reticular formation consists of heterogeneous cell masses rather than forming a homogeneous structure, such activation of individual cells, or at most individual nuclei, would seem more likely than the activation of the whole lateral portion during facilitation and of the whole medial portion during inhibition.

We know that the spontaneous electrical activity of the cerebellum is of two distinct types: high frequency activity at 300 pulses and more per second, and low frequency activity at about 8 to 10 per second. The high frequency activity seems to originate in the cerebellum. It persists when all fiber connections with the rest of the brain are cut and even (for a minute or two) when the cerebellum is removed from the skull. The low frequency activity, on the other hand, seems to result from relays to the cerebellum because it disappears when all connections are cut (Snider and Eldred, 1949).

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* See Nulsen and associates, 1948. Moruzzi later reported that stimulation of the cerebellum at a frequency of 300 pulses per second inhibited postural tone but *facilitated cortical motor responses* (1950, pp. 51–53). Hence the findings of these two groups of investigators are entirely compatible.

† It has been reported that stimulation of the cerebellum, its afferent (brachium pontis and restiform body) or efferent connections (brachium conjunctivum, ventral thalamic nucleus) at 30 volts, 60 per second, can stop electrical seizure activity in the cortex produced by cortical stimulation, but can also facilitate or prolong it (Cooke and Snider, 1955). It is conceivable that the frequency of cerebellar stimulation may either clash with or reinforce the frequency of excitation in the motor area, depending on the frequency dominant there.

Fast electrical activity ("cerebellar-like") appears in the brain stem reticular formation and in the ventrolateral thalamic nucleus (Snider, 1952), and seems to be connected with motor activity. On the other hand, low voltage slow stimulation of various structures has produced inhibition of movements, muscular relaxation, and sleep. Apparently, fast frequency excitation facilitates or induces motor activity via relays to the premotor and motor cortex. The inherent fast frequency beat of the cerebellum seems to *amplify* incoming impulses and so to make action not only possible, but effortless. The dog or cat whose cerebellum has been removed is able to walk, but with considerable effort. He can run a few steps but will do so only when called and encouraged.

Slow frequency impulses seem to be relayed to the cerebellum from the hippocampus and midbrain, and are so organized that they make coordinated movements possible. Every movement demands the contraction of some muscles and the relaxation of others. The circuit relaying such a coordinated pattern to the motor cortex seems to go via the ventral thalamus to make discrete movements possible; and via the basal ganglia to facilitate background movements. Hence, damage anywhere along the circuit from cerebellum to basal ganglia can produce various disorders of movement (tremor, spasticity, ballismus, etc.; see Mettler, 1955) without making deliberate movement impossible.

But over and above the muscular relaxation seen during rest and that necessary for action, both of which imply motor inhibition, there seems to be a kind of inhibition which reduces sensory impressions and vegetative activity. This occurs in sleep and in various states of general relaxation and unconsciousness. We will discuss this general inhibition in connection with sleep and unconsciousness in the next section. For the present, we propose to deal exclusively with the inhibition that is part of action and rest in the waking state, and with the circuit that mediates it.

The inhibitory circuit. McCulloch (1944) has shown that a suppression of movement occurs when impulses reach the caudate and ventrolateral thalamic nuclei; consequently these nuclei must be within the circuit which induces arrest of movement and muscular relaxation. Now we will consider what evidence there is for such an inhibitory circuit.

It is well known that the cerebellum mediates changes in muscular tone.

When it is removed, there is general extensor rigidity. This also occurs after destruction of cerebral and cerebellar suppressor regions—that is, area 24 (or its homologue), the caudate nucleus, the anterior lobe of the cerebellum and the dentate nucleus, and the inhibitory parts of the bulboreticular formation—and is eliminated by spinal transection (Lindsley, 1952). While rigidity consists of exaggerated extensor tonus, spasticity is an exaggerated motor reaction to sensory stimulation. Transient spasticity follows lesions of any of the suppressor areas, but is more marked and persistent when *all* these areas are destroyed. Flexor responses may return to normal in a few weeks, but extensor reflexes may remain exaggerated, appearing as rigidity.

Now it seems that normal tone and movement require an interplay between muscle contraction and relaxation. When a lesion destroys the areas that initiate muscular contraction, or interrupts the circuit mediating it, the muscles will become flaccid—as is the case after isolated destruction of area 4, after pyramidal section, or spinal transection. When those areas are removed that are included in the circuit inducing relaxation, we would expect spasticity and rigidity (as occurs in ablation of area 4s, the suppressor areas in the cerebellum, parts of the caudate and reticular formation).

In the intact brain, the cerebellum seems to relay the impulses both to muscular contraction and muscular relaxation (apparently via the reticular formation) to the thalamus and hypothalamus, the caudate nucleus and premotor cortex. Whether the outcome is to be action or arrest of action depends on the appraisal that induces a desire either to move or to stop moving. However, since crude sensations can be experienced and appraised via the thalamus, and the medial thalamic nuclei are connected with the tectum and tegmentum, a rudimentary appraisal for action seems to be possible for animals below primates even after removal of the cortex. Action can then be executed via the tegmentum, cerebellum, striatum, and the extrapyramidal pathways including the prominent rubrospinal tracts. (In primates, where the rubrospinal tract is inconspicuous, action is impossible after decortication.)

Decorticated animals (except for primates) have normal posture and so do animals decerebrated by a cut above the superior colliculus in the midbrain; for postural reflexes can be activated via the cerebellum, red nucleus, and descending cerebellospinal and rubrospinal tracts. When the connection between the cerebellum and spinal motor neurons is interrupted by eliminating the red nucleus as well, normal postural reflexes become impossible. Now the only system left functioning is the fast frequency system activated by somatosensory influx which excites the vestibulary nucleus and medullary reticular formation connecting with spinal motor pathways; hence there is decerebrate rigidity. When such reflex innervation via the reticular formation is made impossible by a cut below Deiter's nucleus (lateral vestibular nucleus), these motor neurons can no longer be stimulated by sensory afferents and the muscles become flaccid (cf. Lindsley, 1952).

That the caudate nucleus is a relay station in this inhibitory circuit from the cerebellum to the motor cortex seems established from a variety of experimental results. It has been found, for instance, that low frequency stimulation of this nucleus evokes spindles in the electrogram of the thalamus and cortex (Shimamoto and Verzeano, 1954), and such spindles occur typically during drowsiness and light sleep. Heath and Hodes (1952) have actually induced sleep with muscular relaxation by stimulating the caudate nucleus in man and monkey with slow frequency current. There is also evidence that the inhibition of movements is mediated by the lateral part of the caudate nucleus, while the medial portion facilitates movements (Hodes *et al.,* 1951). When only the caudate nucleus is stimulated electrically (without stimulating the motor cortex), there are no movements. This seems to confirm our suggestion that the activation of motor cells requires direct relays from the lateral ventral thalamic nucleus to area 4, as well as relays from the anterior and medial ventral nucleus via the caudate nucleus to the prefrontal cortex, which connects with the motor area. From all these findings we can infer that any excitation of the caudate nucleus can either facilitate movements or inhibit them, depending on the location of the caudate fibers that are excited, or on the stimulation frequency, and perhaps on both.

When the motor suppressor region (area 4s) is painted with strychnine solution, spikes appear in the inhibitory bulbar reticular formation and in the caudate nucleus, while spontaneous potentials in the lateral ventral thalamic nuclei and the motor cortex are suppressed bilaterally. Since sectioning the cortex between areas 4s and 4, or making a ring-like cut around area 4s, or cutting the pyramids does not interfere with these effects, but undercutting of area 4s does (Meyers *et al.,* 1954), the impulse to relax is apparently started from area 4s, travels via the anterior cingulate gyrus (movement appraised as unsuitable), is relayed to the hippocampus, transformed into slow frequency impulses, and relayed via the cerebellum and caudate nucleus to the motor area.

This seems to be confirmed by another experiment, showing that a cut through the midbrain peduncles (which contain the tegmentum) interferes with the appearance of electrical potentials in the bulbar reticular formation after stimulation of area 4s (see Meyers *et al.,* 1954). According to these authors, fibers have been found that connect the caudate nucleus and the motor cortex, but there does not seem to be a fiber connection between the caudate nucleus and area 4s. This would confirm that electrical stimulation of area 4s induces relaxation only via the circuit from the anterior cingulate gyrus to the hippocampus and cerebellum, and the relay to the caudate nucleus and motor cortex.

Our conception of the inhibitory pathway would explain the results reported by Hodes and his associates (Hodes *et al.,* 1951; Peacock and Hodes, 1951; Heath *et al.,* 1954), and explain them in a consistent way. These workers found contralateral inhibition of the knee jerk or of movements produced by cortical stimulation when they stimulated the medial forebrain bundle, the lateral and anterior suprasylvian gyri (homologue of the temporal tip), the lateral half of the caudate nucleus, and the anterior ventral and reticular nuclei of the thalamus. They obtained bilateral inhibition from stimulating the cingulate gyrus and its efferent fibers, the hypothalamus, the medial reticular formation of the brain stem, the midline thalamic nuclei, the septal nuclei, and the mediobasal region of the olfactory and preoptic cortex.

According to our hypothesis, emotional relaxation represents bilateral inhibition, the result of a desire to keep still and enjoy something. Such inhibition could be obtained by stimulating the thalamic and cortical estimative system, or the action circuit anywhere between the hippocampus and cerebellum; and the points from which Hodes and his coworkers obtained bilateral inhibition do fall within these regions. Contralateral inhibition of cortically produced movements could be obtained, we think, by stimulating the second limb of the action circuit after the projection fibers from the cerebellum to the thalamus have crossed over. The medial forebrain bundle, temporal tip, caudate nucleus, anterior ventral and reticular nuclei in the thalamus, which yielded contralateral inhibition, are obviously within this last limb of the action circuit.

There are several structures from which Hodes and associates have obtained both facilitation and inhibition, sometimes from points close together; for instance, from the cingulate gyrus and its radiations, from the septal nuclei, the globus pallidus, the anterior hypothalamus, thalamic midline nuclei, and, of course, from the internal capsule. This would indicate that we are not dealing with separate inhibitory and facilitatory pathways but with patterns of action which inhibit some and excite other motor cells.

Such inhibition over the action circuit seems to influence sensory as well as motor impulses. Electrical stimulation of inhibitory cells in the brain stem reticular formation, for instance, reduces or prevents the sensory discharges of the muscle spindle proprioceptors (Eldred and associates, 1953). A similar inhibition has been reported by Gernandt and Thulin (1955) in the vestibular system, by Granit (1955) in the retina, and by Hagbarth and Kerr (1954) in the olfactory system. In this way, impulses to rest and relax seem to reduce sensory bombardment as well as muscular activity (cf. Ch. 5, p. 159).

SLEEP AND WAKING, CONSCIOUSNESS AND UNCONSCIOUSNESS

We have discussed the alternation of excitation and inhibition in coordinated action, and the partial inhibition of action during rest and relaxation. There is also a general inhibition of action which includes a reduction of sensory experience as well: we call it sleep. A still more profound reduction of activity is found during unconsciousnesss.

We will now examine various states of consciousness and the activity proper to each. We propose to show that there is a change in the activation of the action circuit which accounts for the reduction of activity during sleep. At the same time, we experience some sensations, though not many, during sleep, and engage in the elaborate exercises in imagination that we call dreams. We shall try to discover how the break in consciousness occurs between dream and waking by examining other gaps in memory and other states of unconsciousness.

Conditions of consciousness. Psychologically, we speak of being conscious when we are awake and aware of what goes on around us, when we sense, think, feel, can move and talk, when we can remember our experience and reflect upon it. As Gasson points out:

> What is conscious must indicate some psychological experience, something *lived through* of which we know; a *knowing* or a *wanting* or a *doing* or a

> *happening* in us of which we have direct knowledge and which we refer to ourselves. Such psychological experience, moreover, in itself or in its content, must be fully *available* to us here and now for the needs of the present moment. (1954, p. 209; Gasson's italics)

Thus consciousness includes awareness of the external world and of our own experience present and past.

Even if a man is not able to move or talk, he may be fully aware of what goes on around him. On the other hand, he may move and even speak, yet not be able to reflect on what he is doing, understand or explain his actions, or remember them afterwards. In such cases, there is a falling short of *full* consciousness. But to be conscious even in this imperfect way, he must be able to *perceive* and *appraise* some things at least. And to be fully conscious, he must be aware of the external world and of himself acting in it, must be able to reflect and remember, and to use what he knows.

While the functioning of some afferent sensory pathway is *necessary* for full consciousness, the functioning of the sensory system is not *sufficient* for consciousness. Sensory impressions may be received (as shown by electrical potentials recorded from the sensory cortex), yet a man may be unconscious. To become aware of what our senses receive seems to require something beyond mere sensory activity. It requires the excitation of pathways that belong neither to the sensory nor to the motor system, for a man may be fully conscious though he is completely paralyzed. Awareness seems to be connected with the functioning of a system that links afferent and efferent tracts.

We have described this system in Chapter 3 and have called it the "estimative system." It seems to mediate the appraisal of sensations via its subthalamic and thalamic portion, and of objects and their significance via its cortical (limbic) portion. Unless sensory impressions are felt as good or bad, pleasant or unpleasant, we have no motive to attend to them; and unless objects and situations are evaluated as demanding a particular action, we have no reason either to approach or to avoid them. The functioning of this system allows the *awareness* of what goes on, the *estimate* of how it affects us, and arouses an *impulse to action*. Through its connections with the hippocampus and amygdala, the estimative system allows us to *recall* relevant memories and to *plan* suitable actions; through its connections with the motor system, it allows us to *carry out* the action.

Psychological activity during sleep. The estimative system functions during waking, but it is not completely inactive during sleep. In light sleep and even under light anesthesia we dream, and often have intense dream emotions. We have argued that the experience of emotion requires a circuit from the cortical limbic areas to the hippocampus and cerebellum, and via the thalamus and hypothalamus to the frontal lobe. It seems clear that at least part of this circuit must remain active during dreams.

Let us now examine the difference in our experience during waking and dreaming. In dreams as during waking hours, we live through various situations, we see and hear, think, feel, and act; but when we wake we know we have been dreaming. We did not actually talk or hear others talk, we did not actually see the scenes we dreamed about, we did not actually run or fight or laugh or weep because we have been in bed and asleep. In dreams we do not usually know that we dream, nor do we know that there is a waking life. We like or dislike things and people as if they were real, we are angry and fight, or afraid and try to run away, as if we really were challenged or pursued. When the dream emotion is very intense, as in a nightmare dream, we often try to run yet feel rooted to the spot, or try to cry for help yet cannot utter a sound. Indeed, when immense effort finally does produce a cry, it is often enough to wake us. Even on waking we may go on feeling the dream terror until we realize that there is nothing to be afraid of. Every mother at some time or other has had the experience of trying to wake her child who is screaming in his sleep and is so frightened that she can hardly calm him.

Such reflections seem to indicate that emotions can be experienced during dreams, though the actions to which they impel cannot usually be carried out. Sleepwalking is relatively rare. Apparently, the circuit mediating emotions (and the autonomic changes that go with them) does not activate the motor cortex during sleep. Moreover, dreams occur only during light sleep, on falling asleep or waking up. According to Blake *et al.* (1939), dreams are accompanied by bursts of electrical activity in the EEG, but during deep sleep only slow waves without such bursts can be observed.

A click, a cold touch, or a pistol shot did not arouse a sleeper during the time when the slow pattern of deep sleep could be observed in the EEG, but autonomic changes (psychogalvanic reflex, vasomotor and respiratory changes) were more lively than during waking; repetition did not suppress them during sleep as it did during waking (Jung, 1954). This would indicate that incidental stimuli are experienced during sleep and may even arouse emotion (if that may be inferred from the autonomic reaction which usually accompanies emotion). Now it seems likely that dreams might be occasioned by such incidental impressions which are then woven into dream scenes. Various observations seem to confirm that this actually happens (Beigel, 1960; Ayalla, 1957).

Sense perception in dreams. While awake, we can perceive and appraise an object realistically by drawing upon earlier experience with it. But during sleep there seem to occur merely fleeting isolated experiences, never welded into stable objects which can be appraised according to their earlier effect on us. Of course, only a few sensory avenues are available to the sleeper and this might account for the fragmentary nature of his experience. But it does not account for the fact that his sensory impressions are not referred to the real object but become part of a dream scene. This would imply that the automatic recall of relevant memories which provides the meaning for sensory experience is not possible during sleep.

Psychologically speaking, the recall of memories represents an action impulse: when something seen or heard or touched has to be dealt with in some way, it becomes imperative to discover how it has affected us in the past. During waking hours, each sense impression will automatically arouse an *impulse to recall* similar situations, which will help us to decide on action. Such an impulse to recall would have to be mediated by the hippocampal circuit connecting with midbrain, thalamic association nuclei, and cortical association areas (see Ch. 3).

However, hippocampal activity seems to subside during sleep. Green and Arduini (1943) have shown that the "alert pattern" in the hippocampal electrogram, which consists of slow synchronized waves, becomes desynchronized just before the sleep pattern appears in the cortex. And Thomalske *et al.* (1955) mentioned that the "alert" pattern appeared in the hippocampus just before movement could be observed; hence, they suggested that the hippocampus signals the "intention to move." We might add that it also seems to signal the fact of falling asleep. We have argued that it is the hippocampus that *initiates* action impulses as soon as the situation has been appraised, and it is also the hippocampus that *inhibits* action. When the hippocampus shows the sleep pattern, its activity is sharply reduced, so that incoming sensations can neither be identified nor appraised for appropriate action. Since sense impressions are not identified during sleep, the imaginative activity in dreams seems to use these impressions for the production of dream pictures and dream actions. Of course, as soon as an impression is strong enough to wake the sleeper (by activating the hippocampus, we think), he will interpret the situation correctly and deal with it accordingly. When his bedroom gets cold in the middle of the night, the sleeper may dream that he is a polar explorer suffering from frostbite. But if he feels chilly enough, he will awake and pull up another blanket.

Imagination resistant to inactivation. Imagination is an activity that seems to go on day and night. It is the last to disappear in sleep and goes on during stages of light anesthesia until deepening narcosis blots it out. In a recent investigation of sleep, Dement and Kleitman (1957) found that all their subjects dreamed throughout the night, though not continuously. Stages of deep, dreamless sleep alternated with periods of light sleep and dreaming. We tend to disregard this continuous working of the imagination because we are so engrossed in sense experience that we assume that images come only when we need them for our deliberate purposes. The all too familiar fact that distractions so easily tempt us into daydreams, when we are trying to concentrate on work or studies, should have warned us that images come uncalled even when they are not wanted. Ordinarily, imagination is active continually: whether we think or speak, whether we write a letter or bake a cake, whether we walk to the corner drug store or play golf, we anticipate the next step in the sequence. Head's "body scheme," so necessary for action, is produced by motor imagination; but we also imagine *what may happen,* not only *what we are to do.*

When action is inhibited and sense impressions are missing, we at last become aware of the images that come crowding in spontaneously. Bexton and associates (1954) have reported some interesting observations. When young men were isolated in small cubicles, prevented by goggles from seeing objects, and by cardboard cuffs from touching anything, they experienced vivid visual, auditory, and often somesthetic images. The visual images formed at first simple geometrical patterns, soon progressed to something like wallpaper patterns, next became isolated objects, and finally dream-like scenes. Evidently, the absence of sensory stimulation and the enforced rest allowed such images to appear and become so intense that they finally seemed to appear in space and were experienced as hallucinations.

Emotion directing fantasy. Our fantasy life is never haphazard. Awake or asleep, our emotional preoccupation gives it direction. Whether we work or study, the slightest slackening of interest will bring back a favorite daydream or a host of nagging worries, thoughts of plans to be made or conflicts to be resolved. In the night dream as in the daydream, the emotional attitude or preoccupation will arouse a train of images, picturing things and people and making up actions and action sequences. While asleep, these images are autonomous. They cannot be checked by relevant memories or immediate experience, and the appraisal of what is an appropriate action is strictly in terms of the dream situation. Immediate sense impressions set the stage, but the dream itself is an imaginative play directed by the dreamer's emotional attitudes. When he experiences a particular sensation (hunger, cold, heat, etc.), he seems to appraise it as either pleasant or unpleasant, and to deal with it in the light of his own emotional preoccupation, regardless of its actual significance. A heavy meal late at night may produce unpleasant sensations, but they will develop into a nightmare only if there is some dormant apprehension that allies itself with the experienced unpleasantness. Since he cannot identify his sense experience, the dreamer will picture a situation that might arouse both the unpleasant sensation and his underlying apprehension. If he is haunted by fears of a just retribution for something he has done, he might dream that he has been poisoned by an enemy and is writhing in agony. Thus the dream seems to use the felt organic sensation as a stage prop to express the dreamer's real feelings. The dream emotion is a genuine emotion, tailored to the dreamer's measure, even though the stage setting may seem fanciful or bizarre.

Neurological evidence. To explain the fact that the imagination can function when recall is impossible, we have to assume that relays from the sensory system can touch off the neurons in the limbic cortex and the amygdala at a level of excitation that fails to spread to the hippocampal circuit. Without some appraisal of an experienced sensation, it would be impossible to arouse the dreamer's habitual likes and dislikes (mediated by the limbic region) and to induce relevant visual, auditory, and motor images.

There is some experimental confirmation of this notion. Segundo and associates (1955) found that the superior temporal gyrus, when strychninized, evoked potentials in the amygdala. Prolonged stimulation increased the excitability of the amygdala and, to a lesser extent, of the hippocampus. This seems to show that sensory and association areas can excite the amygdala, and that the hippocampus is not excited as much. Green and Shimamoto (1953) have shown that the sensory excitation that reaches the limbic cortex of the hippocampal gyrus is relayed via intracortical association fibers directly to the amygdala; and Lesse and associates (1955) reported that the electrical activity recorded from the amygdala during imagination did not always spread to the hippocampus. These reports seem to confirm that the amygdaloid complex is excited by sensory impressions without necessarily exciting the hippocampus. There is also evidence of an influx to the amygdala from the thalamic as well as the cortical portion of our "estimative system." According to Pribram, Epstein, and Bagshaw (1952), ablation of the amygdala was followed by degeneration in the midline and intralaminar nuclei and in no other thalamic area.

According to our hypothesis, dreams are products of creative imagination which combines and rearranges memory images. Actually, it has been found that dreams no longer occur when the area necessary for visualization (area 19) is damaged or when the thalamic peduncles are injured (Nielsen, 1941; Gloning and Sternbach, 1953) and neural impulses from the amygdala can no longer reach the visual association area. Dreams also disappear after prefrontal lobotomy. After a year or so, they usually return but in impoverished form (Schindler, 1953). Schindler does not mention what is missing in such postlobotomy dreams, but we would hazard the guess that the dreamer is no longer acting and participating in the dream. To imagine oneself acting seems to require an intact connection between the dorsomedial thalamic nuclei and the prefrontal areas, and this connection is interrupted by lobotomy. We would think that the dreams of lobotomized patients would be reduced to a series of visual and auditory images, much like hypnagogic hallucinations.* It should be rewarding to investigate this problem.

Variations in consciousness and memory. Full waking consciousness gives way to the partial consciousness of dream life and the unconsciousness of deep sleep. Even during waking, full consciousness is often partially submerged or completely blotted out when neural activity becomes paroxysmal, as it does in epilepsy.

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* Dement and Kleitman (1957) found that dreams are invariably associated with the appearance of low voltage, 7 to 10 cycle per second (cps.) waves in the occipital area and 18 to 25 cps. waves in the frontal area. Dreams are also associated with eye movements. And eye movements occur, according to Jacobson (1932), when something is imagined and its position or movement is followed in imagination.

On falling asleep, there were also EEG changes associated with dreams, but no eye movements could be recorded. When roused, the subjects reported not dreams but hypnagogic hallucinations (Dement and Kleitman, 1957). This seems to indicate that the difference between dreams and hypnagogic hallucinations is to be found in the type of imagination. In dreams, movement is imagined in objects and in oneself, while hypnagogic hallucinations consist of purely visual imagery.

In many types of epilepsy, there is a complete loss of consciousness. *Grand mal* attacks are generalized motor fits in which the epileptic falls down and becomes unconscious. In *petit mal* there is a momentary loss of consciousness, a "mental lapse," during which the patient stops what he is doing, has a vacant expression but does not fall. *Dreamy state* seizures are described as a sudden alteration in the patient's consciousness, a sudden feeling of familiarity, or of being far away, or of strangeness, and sometimes also include sudden hallucinatory experiences. *Temporal automatisms* * (Penfield, 1952) or *psychomotor seizures* (Gibbs and Gibbs, 1950) are seizures in which the patient seems to act automatically, without full awareness. This is how Penfield describes such automatisms:

> The patient may exhibit varying degrees or depths of lack of understanding. At the beginning of the attack he may smack his lips and swallow. He may fumble about and he often examines his own clothing in a sort of mechanical self-inspection. The patient may then move about as though he neither heard nor saw and as though he could not speak. A little later he may reply when addressed and may even obey commands momentarily. He may then continue to carry out a project which he had decided upon before his attack, or he may embark upon a repeated performance which has come to be his habit while in such a state. Some patients resent interference vigorously and even dangerously. All patients in this state will have amnesia for the duration of the automatism although they may subsequently remember the sensation or dream that preceded it during the preliminary localized cortical discharge. (1952, p. 522)

We now propose to discuss the psychological state and its neural substrate in the various types of epilepsy.

There are some epileptic attacks, so-called "partial seizures," in which various sensations are experienced (shapes or colors, sounds, smells, tastes, or peculiar organic sensations), but there is neither a loss of consciousness nor a loss of memory. In other attacks, like grand mal and petit mal, there is a loss of both consciousness and memory; and in still others, there seems to be a loss of memory alone.

Psychomotor seizures presuppose at least some perception and appraisal of the real world, and thus awareness, for the patient carries out a particular action successfully. But he seems to be abstracted, caught up in a private world of his own. That he has an imaginative experience of some kind is suggested by the fact that high amplitude spike and slow wave discharges have been recorded from the amygdala, the hippocampus, septal area, and anterior hypothalamus (Heath, 1957); and we know that the amygdaloid complex is active in imagination. Since this is a seizure pattern, the fantasy must be extremely vivid, amounting to a hallucination. Apparently, the patient is acting under duress, forced by the hallucinatory experience which dictates a particular action pattern.

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* These attacks are called "temporal automatisms" because they can be precipitated by stimulation of the anterior part of the temporal lobe, particularly the area around the amygdaloid complex (Feindel and Penfield, 1954).

Though the patient is conscious in the sense that his hallucination seems to fit into the real world and is acted out in it, he cannot recall either the experience itself or his part in it. Ordinarily, recall is not only spontaneous but continuous. At every moment, we recall the moment before and remember both what happened to us and what we did. Because we remember, we are aware of ourselves as persons with a particular history living in a particular place. Thus the memory circuit seems to be active continually during waking, touched off by everything we see or hear, appraise, and want to do. If there should be a pause in this continuous recall, it would mean a momentary lapse of memory. There is such a break during sleep when imagination may be active but recall is suppressed. When dreaming, we do not remember our waking life, though there is an awareness of the dream sequence. This, however, seems to depend on the continuous functioning of the imagination circuit which repeats the sequence it has started. We do not remember one dream when we are caught up in the next, though we may have the same dream again and *recognize* it.

The hallucination induced by a psychomotor seizure seems to create a similar break in the continuity of recall as does the dream. The patient does not seem to remember the job he was about to do or the routine of his daily life. He is living in his fantasy, but the sequence of events in it is dictated by the pattern of spontaneous nervous discharge which spreads from the amygdala to the hippocampus. When the seizure pattern is imposed on the hippocampus, the memory circuit cannot be used for recall. Hence there is as complete a break in the continuity of memory as there is between waking and sleeping. The patient will usually remember the first experience induced by the discharge, the "aura," because the memory circuit is still functioning at that time; but he will be completely unable to recall anything as soon as the discharge has spread to the hippocampus. According to Gloor (1955), the hippocampal response after amygdaloid stimulation has a latency of 25 milliseconds and more, which is ample for experiencing the aura.

This explanation applies to the amnesia for a grand mal or a petit mal attack also. In *grand mal,* the epileptic aura is usually remembered and may give a clue to the part of the neocortex or hippocampus primarily involved. Electrical stimulation of the amygdala does not induce an epileptic attack (Gastaut, 1954), but stimulation of the hippocampus often does (Creutzfeldt and Meyer-Mickeleit, 1953); and cutting the descending fornix columns has reduced the number of grand mal seizures in epileptics (Garcia Bengochea *et al.,* 1954). When the hippocampus is stimulated, the developing seizure may either remain localized in this structure, or it may propagate to the anterior thalamic nucleus and cingulate gyrus, to the amygdala and temporal tip. Or, finally, it may spread to the motor cortex, in which case there is a general convulsion. Hence Creutzfeldt and Meyer-Mickeleit conclude that the hippocampus is the main structure that initiates convulsions. It is conceivable that some grand mal attacks also begin in the hippocampus, either because this structure may be hyperexcitable in some individuals or because such hyperexcitability is induced by convulsants, emotional upsets, or metabolic changes.

That the hippocampus (and through it, the action circuit) is activated by a stimulation of the cortex that produces an epileptic attack can be inferred from a report by Prus (1948). He found that such an attack is not prevented by cutting around the point of stimulation or by cutting the pyramidal or frontotemporo-occipital tracts. But cutting the upper dorsal midbrain, without injuring the pyramidal and pontine tracts, did prevent it. Since the action circuit, as we have described it, goes from the hippocampus via the mammillary bodies and mammillotegmental tract to the brain stem reticular formation and cerebellum, it would be interrupted by such a cut. When Prus applied cocaine to the motor cortex, electrical stimulation of this region produced tonic contractions that stopped when the current stopped, without developing into convulsions. Since cocaine paralyzes receptor endings, it would seem that it prevented the relay of neural impulses to the anterior cingulate gyrus and hippocampus. This would have interrupted the action circuit and prevented the amplification of motor impulses in the cerebellum, and hence the development of convulsions. The electrical stimulation activated only the pyramidal tracts and so merely produced tonic contraction.

Finally, Sano and Malamud (1953) have reported that of fifty institutionalized epileptics, twenty-nine were found to have had sclerosis of the hippocampus and nine had temporal or frontotemporal traumatic lesions. Only five brains showed no degeneration. This would indicate that obvious hippocampal lesions accompanied epilepsy in the majority of cases. In the cases without obvious lesions, the hippocampus may have been hyperexcitable for some other reason.

Petit mal consists of a momentary "absence" without falling and without convulsions. Sometimes the patient has an almost imperceptible twitching of his eyelids or the muscles of his upper arm. After a moment, he continues where he left off and is not aware of his lapse. A similar state may be induced in animals if they are stimulated through electrodes implanted in the intralaminar nuclei. With low frequency and moderate intensity stimulation, there is an "arrest" reaction in which the animal stops its activity and does not respond to stimulation. With more rapid or more intense stimulation, this develops into a typical petit mal attack with slight twitching about the face and eyes. Still more intense stimulation finally produces a grand mal attack, a generalized convulsion (Jasper and Droogleever-Fortuyn, 1947). Matsui (1957) found the same effect on stimulating the medial thalamus in human beings and reported that the induced grand mal and petit mal attacks were identical with spontaneous seizures and showed the same EEG pattern. Apparently, a spontaneous discharge in the estimative system may produce a petit mal attack, but the discharge must be intense enough to spread to the hippocampus before it can induce a general convulsion. Since such a spontaneous discharge prevents the use of the estimative system for appraisal and action, the lapse of consciousness is accounted for. The slight twitching seems to be the result of a spread of excitation, probably to the tectum and tegmentum.

Dreamy states, finally, could be interpreted as mild spontaneous discharges in the association or limbic cortex, which would account for the feelings of strangeness or familiarity and the occasional hallucinations. In this case, recall seems to be continuous so that consciousness is maintained. While our explanations are admittedly hypothetical, even speculative, they fit in with our previous analysis and provide a framework for further investigation.

Part II. THE PSYCHO-PHYSIOLOGICAL STATE

5. EMOTION, CONSCIOUSNESS, AND THE EEG

The investigation of brain function during various states of consciousness has made great strides since the discovery that the brain shows continual changes in electrical potential throughout its volume, which can be recorded by means of scalp electrodes. These "brain waves," as they have been called, indicate primarily the electrical activity on the surface of the cortex. However, implanted electrodes have been used in recent years to record electrical potential changes from deeper layers of the cortex and from subcortical structures. We have had occasion to refer to evidence from these studies throughout the last three chapters.

Now we propose to review briefly the relevant knowledge we have gained from these brain wave records, the electroencephalogram (EEG), and to discuss two systems in the brain that have been found to influence its activity: the ***brain stem reticular system*** and the ***diffuse thalamic system***. Stimulation of these systems, either during sensory experience or by an electric current, produces characteristic EEG changes, so-called "EEG activation"; hence they are called "activating systems."

Next we shall review the evidence that led to the notion of two separate though connected activating systems and shall see how these systems are related to the theory of brain function we have discussed here. We hope to show that both the ascending brain stem reticular system and the diffuse thalamic system are part of what we have called the estimative system; and also, that the descending part of the brain stem reticular system is part of the action circuit we have described.

THE ELECTROENCEPHALOGRAM

Hans Berger demonstrated in 1929 that there are continual changes in electrical potential in the living brain which can be picked up and recorded with suitable amplification. It has been found that the voltages of these potential changes range between 100 and 1,000 microvolts on the surface of the cortex but are only one tenth to one twentieth of this voltage when recorded through skull and scalp.

These brain waves occur in the cortex even without afferent stimulation, probably as the result of conduction in closed circuits. That such conduction includes subcortical structures is shown by the fact that a cortical area, isolated by a ring-like cut from the rest of the cortex, shows unchanged electrical activity until it is separated from subcortical regions by undercutting (see Wright *et al.,* 1954).*

Brain waves represent electrical potentials from large aggregates of cells which combine and interact. They are recorded from a given brain area through leads attached to the scalp, and appear as a series of simple wave patterns, even though each wave represents the sum of potentials from thousands of individual cells and fibers. They may be recorded from man as well as from animals and show typical patterns for sleeping and waking. Some of these patterns have been identified and studied extensively. They can be specified in terms of the frequency, amplitude, and contour of the individual waves.

The voltage, as shown by the amplitude of the deflections, differs in different individuals and may range from zero to fifty microvolts. Usually, the amplitude of the EEG is given as "low," "average," or "high."

The alpha rhythm. The human EEG shows a dominant pattern when an individual is at rest, with his eyes closed, but not asleep. This is the *alpha* pattern, consisting of a regular series of rather smooth waves of about 7 cycles per second (cps.) frequency. Alpha waves are recorded primarily from the occipital cortex but may also come from parietal and frontal areas. When the eyes are opened or attention is otherwise engaged (e.g., in mental arithmetic), the occipital alpha rhythm disappears. According to Walter (1953), this is true only for people who are predominantly visualizers. Those who use more auditory or kinesthetic images in thinking show an uninterrupted alpha rhythm even with eyes open or during mental work.

Other rhythms. In the frontal cortex, a faster rhythm, the *beta rhythm,* with frequencies between 18 and 32 cps., is often found simultaneously with the alpha rhythm, but more frequently only beta rhythm, together with a still faster frequency (35–55 cps., the *gamma rhythm*), is recorded from the frontal areas during waking. The amplitude of gamma waves is very small. With insufficient amplification, they are sometimes not seen at all. Originally, all waves slower than alpha were called *delta waves*, but more recently waves of 4 to 7 cps. have been called *theta waves* (Fig. 9).

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* However, as long as there is any chemical or mechanical stimulation (e.g., via an intact blood supply), electrical activity will be found in the isolated cortex (Jasper and Van Buren, 1953).

These wave patterns are not fixed but change continually, according to

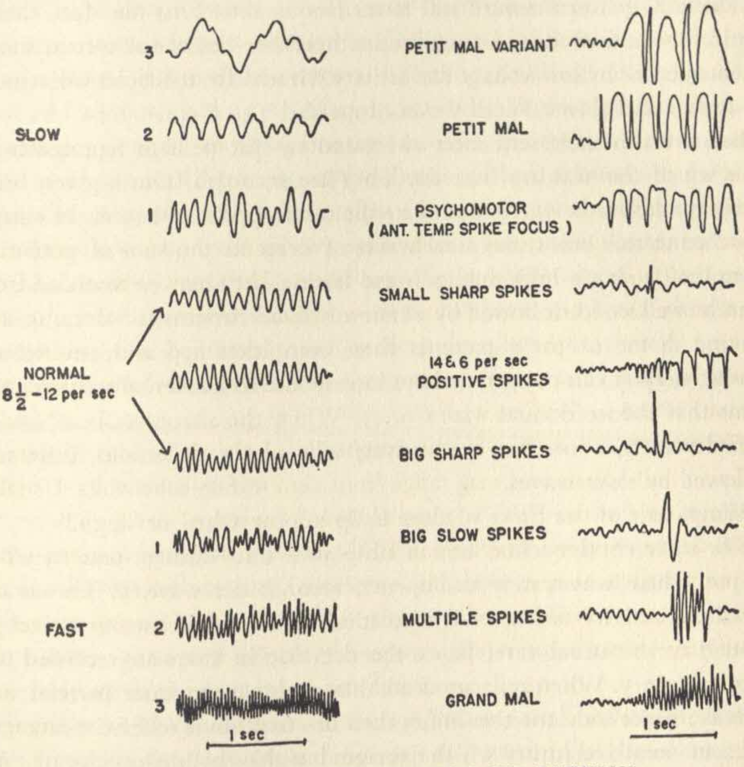

FIGURE 9. NORMAL AND ABNORMAL EEG PATTERNS

In the left column are shown the nonparoxysmal patterns. Above are waves slower than alpha and below, those faster than alpha. In the middle are patterns that fall in the 8½ to 12 cycles per second range, which is the normal alpha range for adults. The right-hand patterns show various types of abnormality. The pattern characteristic for psychomotor seizures is seen over the entire cortex during a clinical seizure; in seizure-free intervals, it appears as an anterior temporal spike focus.

Source: Gibbs and Gibbs. 1951. Atlas of Electroencephalography. Cambridge, Mass., Addison-Wesley.

the state of brain activity. With sensory stimulation or excitement of any kind, higher frequencies tend to replace the lower frequency alpha waves. This has been called "alpha blocking" or "EEG activation." With increasing relaxation, on the other hand, lower frequency waves with increasing amplitude become dominant; hence the trains of alpha waves reappear. When the alert but relaxed state merges into sleep, there is a further change in the EEG pattern toward still lower frequencies. First the alpha waves begin to wax and wane in amplitude, then they become slower and may be interrupted by low voltage fast activity. Gradually, the alpha frequencies disappear and slow smooth waves of around 3 to 6 cps. can be observed. There are also short sequences of around 14 cps., perhaps representing a slowing of the waking beta rhythm (Jasper, 1941). During very deep sleep, random slow waves dominate the tracing. When the sleeper's name is called at this point, the slow waves of deep sleep give way to the "K complex," a single high voltage wave lasting between one sixth and one fourth of a second, followed by a train of higher frequency waves, usually around 14 to 16 cps. Sometimes these complexes appear spontaneously during sleep (Cohn, 1949), perhaps in response to incidental sense impressions that induce dreams (see Ch. 4). When the sleeper wakens, the K complex merges into fast beta waves; when he goes back to sleep, it is followed by slow waves.

Waves as slow as those of deep sleep appear when nerve cells are injured, in cerebral edema, near a neoplasm, and during some types of stupor. These waves may last up to 2 seconds per wave. When normal metabolic activity is hindered in cortical cells, they cannot generate potentials at the usual rate; hence the decrease in frequency around any focus of injury. When cells are dead, they no longer produce any electrical activity; hence a destructive injury does not contribute to EEG potentials. An acute localized injury which damages but does not destroy the neurons produces a well-defined focus of delta waves; but a slowly growing tumor may not reveal any electrical abnormality for a long time because the surrounding tissue is apparently able to keep functioning.

Within limits, frequency decreases as amplitude (voltage) increases. High voltage discharges are the result of very large numbers of neurons discharging together in unison (hypersynchrony). Excessive hypersynchrony (with voltages of 500 to 1,000 microvolts) is associated with epileptic seizures. An epileptic seizure may be ushered in by high amplitude alpha or beta waves which merge into paroxysmal high voltage patterns (cf. Fig. 9). The contour of the wave, that is, the form of the recorded oscillation in electrical potential, may be sharp (a spike), square, serrated, sinusoidal, or so complicated as to defy description. The contour, like the frequency, may change in different states of excitation.

Development of the electrical activity in the brain. The only electrical activity observed at birth is in the central (sensorimotor) region. From here, 14 cps. sleep spindles can be recorded when the infant is asleep, 3 to 6 cps. waves when he is drowsy. There are also 7 to 8 cps. rhythms which persist, with a slight increase after one year of age, until the adult frequencies are reached. About the time visual functions mature, at around three or four months of age (according to Lindsley, 1938), waves of 3 to 4 cps. can be recorded from the occipital area. This frequency increases to 4 to 5 cps. at six months of age and to 4 to 6 cps. at nine months. By the time the infant is one year old, the frequency in the occipital area is usually 6 to 7 cps., and increases gradually until the adult alpha frequency is reached. During childhood, the slower rhythms gradually disappear, though they may be observed during sleep. In adults, the alpha frequency varies between 8 and 13 cps. but decreases in old age. The frequency of the alpha rhythm for any one individual is remarkably constant during any one period in his life. The frequency of the beta rhythm usually increases with advancing age.

It seems that increased excitation, whether it is the result of increased stimulation or increased internal activity, results in faster waves of lower amplitude. But with extreme paroxysmal excitation, as in epilepsy, amplitude increases sharply with only a slight decrease in frequency. On the other hand, in states of depressed function, frequency decreases markedly with only moderate increase in amplitude.

Significance of the EEG. Now the question arises: What is the psychological significance of the EEG? Or, put differently: Can the activity indicated by the various patterns of potential changes be identified with any type of psychological activity?

To answer this question, we must first turn to current explanations of how the potential changes recorded in the EEG are produced. There seems to be general agreement that these changes are the result of processes of polarization and depolarization of cortical neurons, but investigators disagree as to what causes these processes. Some theorists hold that both processes are the result of metabolic changes in the neurons. Others think that polarization is the result of some metabolic change, but that depolarization occurs when volleys of incoming action potentials (derived from neural excitation) trigger off the stored electrical charge. Or depolarization may occur spontaneously, when the charge of the neuron exceeds a critical level so that any factor capable of generating potential differences will touch it off (Cohn, 1949).

Recent evidence seems to favor the view that the potential changes recorded as brain waves are induced by excitation of neural pathways. The neuronal discharge produced by therapeutic electroshock, for instance, is recorded as high amplitude, fast frequency activity in the EEG, which is identical with that occurring in an epileptic convulsion. Moreover, the same frequencies observed in the EEG with scalp electrodes are also recorded from electrodes placed in deeper strata of the cortex. Indeed, the usual EEG activation (alpha blocking and fast beta activity) has been produced after a light flash and recorded in the optic radiation before it appeared on the surface of the occipital cortex (Sem-Jacobsen *et al.,* 1953).

It is usually supposed that the depolarization of large cell masses results in a uniform beat which will appear in the EEG as long trains of regular synchronized waves. These may be alpha trains or the slow waves of sleep, or the high amplitude, fast frequency discharges that occur during an epileptic seizure. By the same token, it is assumed that in normal neuronal excitation the stimulated cells discharge individually or, rather, in comparatively small assemblies; hence, many potential differences cancel out and only small and irregular deflections appear in the EEG.

Epilepsy and the EEG. Thus far, the EEG has been used primarily for the diagnosis of tumors, epilepsy, and brain damage, all of which alter the rhythmic beat of the resting brain. Many of these changes tend to disappear when the patient is alert and attentive, or engrossed in some problem. During such activity, the fast rhythms either mask spontaneous neuronal discharges or perhaps abort them before they have reached seizure intensity. To arrive at a diagnosis in the absence of epileptic seizures, the EEG is recorded when the patient is at rest and has his eyes closed; it is also recorded during drowsiness and sleep.

Grand mal attacks are associated with extremely fast high amplitude synchronized deflections, *psychomotor attacks* with spike and slow wave activity which starts in the anterior temporal lobe, and *petit mal* with a spike and wave discharge which is usually bilateral and most evident in the frontal areas. According to Gibbs and Gibbs (1946), the typical high voltage seizure discharges are greatly increased during sleep and the pattern stands out more sharply. However, most of the patterns typical for epilepsy tend to appear in the first twenty minutes of sleep, disappear during deep sleep, and reappear again shortly before waking. An outright grand mal convulsion can sometimes be precipitated by waking the patient when a typical epileptic pattern is recorded in his EEG; but if he is allowed to sleep on, there is usually no convulsion despite the EEG seizure pattern. Accordingly, these authors suggest that the cortex is not only de-afferented during sleep, as is usually supposed, but also de-efferented, because a seizure pattern in the EEG, which would result in convulsions in the waking state, shows no detectable motor symptoms during sleep. Psychomotor attacks, like grand mal seizures, are comparatively rare—about one to four a month—so an EEG may be taken at a time when there is no sign of seizure activity. According to Gibbs and Gibbs, all seizure discharges are increased during sleep and the diagnosis is facilitated if the EEG is recorded during sleep as well as during waking. Ninety-five percent of patients with psychomotor epilepsy, who showed no seizure activity in their EEG when awake, did show such patterns during sleep. Petit mal attacks, on the other hand, occur many times a day and show thousands of typical discharges in the EEG; thus the waking EEG is sufficient for diagnosis.

The absence of convulsions during sleep, though there are seizure discharges in the EEG, seems to indicate that the motor circuits must be active before a convulsion can occur; and the motor circuits are activated, we have argued, when neural impulses can be relayed from the hippocampus to the brain stem, cerebellum, and frontal lobes. It seems that sleep favors the appearance of electrical signs of an epileptic attack but does not provide favorable conditions for an actual convulsion. This can be explained if we assume that the epileptic focus in these cases is in or near the neocortex (so that scalp electrodes will pick up the discharge) but that a grand mal convulsion can occur only when the hippocampus is triggered off. In Chapter 4 we have given some evidence for this hypothesis. During waking hours, the ceaseless electrical activity in all the circuits may abort the discharge in individual neurons, so that there is but rarely a large enough neuron pool to trigger off the hippocampus. During sleep, when seizure activity in the epileptic focus can develop, the hippocampus is depressed and cannot relay the excitation.

The normal EEG. The EEG is normal when it shows a variation of not more than 2.5 cps. in different combinations around the alpha frequency. It may also show very isolated slower waves (up to 7 cps.) in frontal, parietal, and temporal leads, and prominent fast activity (about 20 cps.) in frontal leads, as long as these waves do not occur in high amplitude volleys. Moderate high voltage activity (between 50 and 70 microvolts) may appear in parieto-occipital leads, as long as it does not occur in bursts. The EEG becomes abnormal when the alpha range varies by more than 2.5 cps., when slow waves (under 8 cps.) appear more frequently, when the voltage is very high (over 100 microvolts) and amplitude modulation is irregular, when slow activity dominates the tracing, when there are high voltage fast volleys or spike and slow wave complexes (Cohn, 1949).

Such a classification of normal and abnormal electroencephalograms takes account of the gross abnormalities found in brain damage or epilepsy, but does not distinguish between what is clinically normal and abnormal. Cohn (1949), for instance, using a control group of 251 healthy Navy men, found "abnormal" tracings in 15 percent, in spite of the fact that no clinical abnormalities could be detected and less than 5 percent of the whole group had been ill from any cause during their tour of duty. Thus it is admitted that an "abnormal" EEG does not necessarily indicate clinical abnormality and, conversely, that a "normal" EEG may be found in clinically abnormal states.

THE EEG AND EMOTION

The normal/abnormal classification of the EEG is obviously strictly empirical and not based upon any understanding of the factors that produce the varying types of wave form, frequency, and voltage. There have been repeated attempts, however, to correlate at least some of the prominent wave patterns with psychological functions.

The pattern investigated first was the alpha rhythm. It was soon found that sensory stimulation, particularly visual stimulation, blocks the alpha rhythm and induces fast beta rhythms in occipital and parietal leads, as well as in other sensory areas (Lindsley, 1951). The same "activation" occurs during attention, mental effort, visual imagination, and also in emotion (Lindsley, 1951; Friedl, 1954). During apprehension, the beta rhythm increases—in anxiety neurotics it predominates markedly when compared with the records of normals (Cohn, 1949; Kennard, 1953).

Lindsley's "activation theory." On the basis of these facts, Lindsley suggested that fast rhythms in the EEG indicate the extent of neural excitation. Since emotion is accompanied by intense EEG activation, he suggested that emotion represents the extreme of neural excitation. EEG activation is induced by stimulation of the brain stem reticular formation and permanently abolished by lesions in this area. Hence Lindsley postulated that it is this system which is responsible not only for EEG activation but for emotional excitation. On the strength of the connection between the brain stem reticular formation and the diffuse thalamic system, Lindsley agreed with Ranson and Magoun's (1939) suggestion that stimulation of the rostral end of the activating system in the hypothalamus may discharge upward into the thalamus and cortex as well as downward toward the periphery and so give rise to the experience of emotion as well as to emotional expression.

Psychologically, Lindsley's theory is a variation of Duffy's concept of "energy mobilization" (Duffy, 1934, 1951; see also Vol. I, Ch. 8), except that the vague concept of "energy" is now replaced by neural excitation of the brain stem reticular formation which induces the arousal effect in the EEG. Lindsley's theory, like Duffy's, assumes that all activity is arranged along one continuum of which one pole is the apathy of stupor or deep sleep, the other intense emotional excitement. The objection to Lindsley's theory is basically the same as to Duffy's: in such a continuum there is no qualitative difference between emotions, nor can emotion be distinguished from effort. There is some evidence that not all emotions induce the fast waves of EEG activation: slow theta waves are commonly found in adolescents with behavior disorders and in aggressive psychopaths (Hill, 1952; Jarvie, 1953), and according to Walter (1950), the appearance of a theta rhythm is a sign of bad temper.

There is no doubt that the activation of the EEG, expressed in blocking of the alpha rhythm and increased beta activity, occurs in emotion; but it also occurs in other states of activity. It seems established also that the brain stem reticular formation is active during such arousal, but it may merely relay neural excitation, not generate it. Altogether, the connection between emotion and EEG activation, between the brain stem reticular formation and states of activity, is more complicated than Lindsley's theory would indicate, a fact admitted by Lindsley himself when he says: "The activation theory appears to account for the extremes but leaves intermediate and mixed states relatively unexplained as yet" (1951, p. 509).

In recent years studies on conscious men and animals, with electrodes embedded in various brain structures, have shown that the EEG is a pale and doubtful reflection of the activity going on in the different structures of the brain. Only when such activity is intense or prolonged does it influence the EEG. In fact, electrical discharges of seizure intensity may occur in some parts of the brain (e.g., in the limbic system or the amygdala) without much change in the EEG, as happens sometimes in psychomotor epilepsy (Brazier, 1954; Heath, 1958). For this reason, it seems unwise to attempt to relate psychological activity to the EEG until the circuits connecting cortical with subcortical areas have been securely established. So doing might make it possible to infer the functional significance of EEG patterns.

Darrow's theory. Darrow (1947, 1950) maintains that the alpha rhythm indicates autonomic (sympathetic) regulation of cortical activity. In cortical excitation, the stimulated cells discharge individually, potential differences are summated algebraically, and only small deflections remain, resulting in the low voltage fast activity seen in the EEG during waking mental activity. Darrow thinks that alpha activity, which represents great masses of neurons beating in unison, creating large potential differences, is an indication that sympathetic activity has cut short the cortical excitation. The activity of the sympathetic nervous system, according to Darrow, constricts cerebral blood vessels, reduces the acetylcholine content of the brain, and so produces alpha activity. Darrow and his coworkers have found in earlier experiments that slow waves in the EEG are accompanied and probably produced by the constriction of blood vessels in the brain. Thus he conceives of the alpha rhythm not only as recovery after cortical excitation but as a positive inhibition.

For Darrow, fast electrical activity in the brain represents inadequate regulation by the sympathetic nervous system, and therefore persistent cortical excitation. When this happens, cortical activity may become self-perpetuating through a kind of feedback effect and give rise to "cortical tension" or anxiety. In emotion, the opposite seems to happen: the over-regulation by the sympathetic nervous system results in reduced cortical activity with increased alpha and theta frequencies.

Among other evidence, Darrow mentions the case of a boy who had frequent outbursts of rage and showed an EEG with strong 7 cps. ("alpha-like") bursts in frontal and motor areas. After the boy had been given prostigmine (which preserves the parasympathetic mediator acetylcholine) for eight days, his emotional outbursts subsided and so did the slow wave pattern. As a check, he was then given atropine (which prevents parasympathetic effects) and the slow EEG activity together with the outbursts of rage returned. Darrow concludes that prostigmine counteracted sympathetic overregulation which had been responsible for the emotional outbursts. Atropine, on the contrary, allowed sympathetic activity to dominate and so reinstated the emotion.

However, prostigmine, like acetylcholine itself, has not only peripheral parasympathetic effects but also produces central effects, namely, an EEG arousal in the resting animal, probably by enhancing the diffuse projection to the cortex. Atropine not only prevents peripheral parasympathetic activity but also seems to prevent the conduction of nerve impulses via the diffuse thalamic system to the cortex. We shall see later in this chapter that this diffuse projection makes it possible to reduce cortical irritability or to increase it. When atropine prevents such control, sensory experience is bound to be more intense and may easily arouse emotion.

That increased alpha and theta rhythm is not synonymous with sympathetic dominance is shown by the fact that it is most consistently observed in patients with low blood pressure (Darrow, 1950); yet chronic sympathetic overactivity is associated with high blood pressure. According to Darrow's theory, patients with sympathetic overactivity should show decreased beta and increased slower patterns; yet Cohn (1949) reports that fast beta activity is characteristic for patients with high blood pressure. Darrow's report that vasoconstriction in the brain goes with increased alpha and slower rhythms is not confirmed by others (Hill and Parr, 1950). This also urges to caution.

Darrow distinguishes between *anxiety* ("cortical tension"), which is accompanied by fast activity in the EEG (deficient sympathetic regulation), and *emotion,* accompanied by slower patterns (sympathetic overregulation). But there is no reasonable basis for such an opposition between anxiety and emotion. On the contrary, anxiety has been considered an emotional state by most theorists. Lindsley even takes it as the prototype of emotion, and concludes from the fact that fast EEG activity (particularly in frontal regions) is associated with anxiety that all emotions must represent EEG activation. He thinks that the slow rhythms reported by some investigators may be based on artifacts in recording.

From the evidence it would seem that emotions may be associated with either fast or slow rhythms. In some emotions (fear, apprehension, anxiety) there is a fast beta pattern (EEG activation), which can be duplicated by adrenaline injection (see below). In others (anger, bad temper) slower rhythms have been observed.

In later publications, Darrow (1952, 1953) has reported that stimulation and EEG activation are followed by increased anterior dominance of alpha-like activity in motor and frontal areas of emotionally unstable and young people and also of brain-injured patients. In well-adjusted adults, on the other hand, stimulation is followed by posterior dominance of these rhythms. He thinks that precentral alpha dominance indicates beginning motor activity, while postcentral dominance may preserve the new motor adjustment. We shall let Darrow and his associates speak for themselves:

> The recorded alpha activity is not itself the projection process nor does it provide integration of the complex cortical and subcortical patterns. It is only the cortical-subcortical liaison for somesthetic control. If, on the one hand, a dynamogenic subcortical urge becomes strong enough, the projected precentral rhythm will dominate the precentral cortex, and the rhythm, when fed back to the subcortex may release the appropriate efferent discharge to the spinal cord. This provides for subcortical drives to action. If, on the other hand, a postcentral somesthetic postural adjustment becomes unsatisfactory, a decrease of postcentral rhythm may allow spread and increased dominance by the out-of-phase precentral rhythm and permit pending motor adjustment. This provides for cortical release (voluntary control?) of action. (1952, p. 17)

Thus Darrow distinguishes between action forced by emotion and action carried out intentionally. In emotionally impelled action, he conceives that the electrical activity induced by action continues (frontal alpha dominance), while in deliberate action it seems to subside (postcentral alpha dominance). These differences may be important, but as long as the origin of alpha waves and their psychological significance is in doubt, it seems premature to draw conclusions from restricted evidence.

We shall attempt to come to some understanding of the EEG after we have summarized recent findings on the two systems which induce such marked changes in the electrical activity of the brain that we might expect to discover something about the EEG patterns from a discussion of their function. Those systems are the brain stem reticular formation and the diffuse thalamic projection system.

BRAIN FUNCTION DURING SLEEP AND WAKING

We have seen that there is a difference in the EEG pattern between sleep and waking, which should indicate some difference in brain function. During waking, there seems to be continuous activity over the action circuit (discussed in the last three chapters), which would account for the fast frequency waves seen particularly in frontal areas. During sleep, this circuit seems to be largely inhibited, and fast waves drop out.

But sleep is more than merely an absence of motor activity. Sensory experience also is reduced or inhibited, though sensory potentials can be recorded from cortical areas during sleep as well or better than during waking. During sleep, sensory impulses are received, but they do not always wake the sleeper. Even strong stimuli will have no effect during the first deep sleep after long vigil. Vegetative functions also are reduced: respiration is slowed, there is a slight fall in temperature and blood pressure, and liver function declines (see Hess and Akert, 1950). The basal metabolic rate drops and various reflexes are depressed or abolished (Peele, 1954).

Such a state of reduced functioning seems to imply generalized inhibition. We have seen (Ch. 4) that some muscles are always inhibited during waking because coordinated action requires the excitation of some neurons and the inhibition of others. We have suggested that resting after action implies the relay of slow frequency excitation to many motor neurons, so inhibiting them. And a general relaxation of voluntary muscles is a recognized technique for inducing sleep (Jacobson, 1929). The technique is successful because a man's attention is directed to various muscle groups, one after another, to imagine relaxation and so to achieve it. Being fully engaged in relaxing his muscles, he cannot use his imagination to picture things that might worry or excite him and thus keep him awake.

The diffuse thalamic system. To succeed in a general damping of activity, the circuit mediating action and rest must be connected with structures that have a widespread projection to cortical and subcortical areas and can reduce the activity of the brain to the point where the alpha waves of the resting state give way to the spindles of light sleep and finally to the slow waves of deep sleep. The structures of the diffuse thalamic system have such a widespread projection. They include the intralaminar nuclei and the nuclei of the midline. This system is connected with the caudate nucleus, the cerebellum, and other subcortical regions, and projects diffusely to all areas of the cortex via the thalamic reticular nuclei (Hanbery *et al.*, 1954; see Fig. 10). The reticular nucleus straddles the fibers of the internal capsule like a crescent comb. Its axons end in the superficial layer of the cortex where they can affect the dendrites of the pyramidal cells in the deeper layers (Jasper, 1954).

When this system is stimulated by a slow frequency current, there is a train of 8 to 10 cps. spindles in the EEG, seen after an appreciable delay, which has been described as the "recruiting response" by Forbes and others (see Brazier, 1954). It seems to occur when more and more neurons are recruited by the stimulating current and induced to conduct at the same frequency. The recruiting response does not occur during waking and can only be recorded when the animal is under light anesthesia. At the same time, this phenomenon is a response to stimulation, and thus seems to indicate a more active state than that during sleep or under anesthesia. This seems confirmed by the fact that it is similar to the alpha rhythm or to sleep "spindles" (Brazier, 1954). Forbes also noticed that the recruiting response shows the same form as the spontaneous bursts of 8 per second waves that alternate with electrical silence in EEG records taken under barbiturate anesthesia. When anesthesia deepens, the frequency slows to 3 per second, similar to the large slow waves of deep sleep.

That the recruiting response is intimately connected with the nuclei of the diffuse thalamic system was shown by Shimamoto and Verzeano (1954) who stimulated the caudate nucleus with single shocks and obtained 8 to 10 per second spindle bursts both in the thalamus and in the cortex after an interval of 166 to 277 milliseconds. Such bursts disappeared in the cortex when the nuclei of the diffuse thalamic system were destroyed, but could be seen in the thalamus after cortical destruction. This seems to indicate that the nuclei of the diffuse system must be intact if this response is to appear in the cortex; but it does not necessarily show that the response originates in the diffuse system. The latency is long enough to allow for a circuit via other subcortical structures.

Slow frequency (or low intensity) stimulation of the diffuse system induces sleep in the waking cat, while higher frequency or intensity arouses the animal from sleep (Hess, in: Delafresnaye, 1954). This has been confirmed for the rat. Stimulating medial thalamic areas in the waking rat with low frequency, low intensity current, Caspers and Winkel (1954) were able to induce sleep with slowing of the EEG, reduction and finally cessation of overt activity, and a depression of vegetative functions. When the same points were stimulated with higher frequency current, or at higher voltages, the rat began to wake up and move. Simultaneously, waking patterns appeared in the EEG. Sleep has been induced in waking human beings by stimulating the medial thalamus at 12 volts, 3 cps. for two to three minutes. From the same points stimulation produced arousal, EEG activation, petit mal, and finally grand mal attacks, as the stimulation frequency was increased (Matsui, 1957).

Diffuse cortical control. It seems that the diffuse thalamic system, through its projections to the cortex, can either increase the excitability of the cortex and action circuit or reduce it. It seems to inhibit motor cells and depress sensory and association areas, probably by a hyperpolarization of the neurons which makes them temporarily inexcitable (Eccles, 1953). As more and more neurons are included in the "recruiting response," movements will become slower, attention will decrease, sensory impressions will be experienced dimly if at all, and a state of drowsiness will eventually merge into sleep.

Psychologically speaking, sleep seems to come when a man becomes indifferent to sense impressions and even detached from them. If the intralaminar and midline thalamic nuclei mediate the appraisal of sense impressions, as we have suggested, slow conduction in this part of the estimative system would make it difficult to appraise what is experienced and lead to indifference. Moreover, when the estimative system begins to conduct at a rate that is too slow to excite the hippocampus, the memory circuit cannot function and the imagination alone is left to interpret sense impressions and weave them into dreams, until the gradual slowing of conduction finally prevents even the excitation of the amygdala. At this point, the EEG would show the slow waves of deep sleep.

The conduction of impulses in the estimative system may be slowed because of synaptic fatigue or drugs that delay transmission. Brazier (1954), for instance, has found that anesthetics act primarily by depressing synaptic transmission, though the conduction of impulses over nerve fibers is delayed as well. We know that synapses are more susceptible to fatigue * than nerve and muscle fibers, and in fatigue synaptic transmission is de-

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* Whether fatigue is eventually found to result from the accumulation of metabolic products which depress synapses, or from any other cause, is immaterial to our argument.

Incidentally, such synaptic fatigue and consequent reduction in the speed of pressed also. The hippocampal circuit (mediating memory) includes more synapses than the amygdalar circuit (mediating imagination), and the action circuit has more synapses than either. This would account for the fact that action drops out first when sleep is about to overcome a man, next memory, and, last of all, imagination.

Nervous conduction may also be slowed by a deliberate decision to relax, or by the relaxation brought about in the induction of hypnosis. An intention to relax, mediated by the hippocampus, could excite slow frequency relays from the cerebellum to the caudate and ventral thalamic nuclei, and also to the nuclei of the "diffuse (estimative) system." Since this system is connected with the whole cortex, it can gradually recruit more and more cortical cells and so reduce cortical excitability. This will result in an appraisal that any action or the recall of relevant memories is difficult—which would renew the intention to relax. Eventually, most of the hippocampal circuits will conduct at reduced frequencies and psychological activity will come to a standstill. When sufficient circuits have been recruited in this way, the slow frequency conduction will appear as slow waves in the EEG.

This interpretation seems supported by the fact that recruiting responses can be induced by stimulation of the cingulate gyrus and of the hippocampus, not only by stimulation of the caudate nucleus (Jung, in: Hess and Akert, 1950). We have suggested that the cingulate gyrus mediates the appraisal of action. When movements have slowed down or become effortful, action is felt as tiring and difficult (via the cingulate gyrus) and will be arrested (via the hippocampus) until most action systems have been inhibited and man or animal is ready for sleep. According to Hess (in: Hess and Akert, 1950), the functioning of various systems is gradually reduced during sleep, though this reduction is never simultaneous in all systems, nor does it happen in an unchanging sequence.

Sleep may be general or may affect particularly the motor or the sensory system. Hess mentions the difference, for instance, between a cat that is asleep in a comfortable position, yet opens its eyes at the slightest sound, and a cat that is asleep sitting up with head hanging down, yet does not really wake, even if there is considerable noise. Occasionally, muscular contractions or movements may go on during sleep. There have been in-

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nervous transmission is also involved in "mental fatigue." To say that such fatigue is "subjective" because oxygen consumption during mental work is negligible, is to misunderstand the problem completely.

stances of men sleeping while standing guard or even marching. But these actions were begun in the waking state: the appraisal that they had to do this originated a continuing action impulse (a "set" organized in the cerebellum) which was never countermanded by another appraisal or a decision for sleep. Under such conditions, sleep comes in snatches punctuated by desperate efforts to keep awake. Apparently, slow frequency conduction in the estimative system prevents memory and recall, or even the appraisal of the present situation. But there is still an awareness that the ongoing action is appropriate and to be maintained. When fatigue is great or the decision to remain awake breaks down, sleep will gradually deepen until progressive muscular relaxation makes it impossible to resist sleep. Until this happens, a restricted action circuit seems to continue functioning, in much the same way that restricted muscular contractions can continue even in profound sleep, i.e., in sleeping with the eyes closed, which requires muscular contractions.

Even though we are suggesting that an appraisal can arrest action and arouse an impulse to lie down and rest, this does not mean that sleep can be induced at will. We can lie down instead of moving about, but as long as we keep on thinking, planning, and worrying, we keep enough action circuits open to prevent sleep. Our decision can provide the external conditions for rest and sleep, but unless we have learned to inhibit not only the impulse to move about but also the impulse to imagine the actions we want or ought to take, sleep will elude us. Instead, we may find ourselves more and more wakeful, urged by this or that emotion.

The ascending reticular system. Some years ago, Magoun and his associates reported that electrical stimulation of a region which includes the reticular formation and tegmentum of the lower brain stem (approximately from the lower pons to the subthalamus, dorsal hypothalamus, and ventromedial thalamus) induces EEG activation, similar to that resulting from sensory stimulation. They suggested that this region maintains the waking state. The "alpha blocking" during such EEG activation, together with the appearance of fast waves, occurs particularly over the frontal region and endures for long periods after stimulation.

In a series of studies, summarized by Magoun (1954), this system was explored further. It was found that sensory stimulation relays impulses over the same units in the ascending reticular system that can be activated by stimulating electrodes implanted in the brain stem reticular formation; that the activity in this region is relayed to the cortex via a thalamic and an extrathalamic pathway; that damage of the rostral portion of this system results in coma, and slightly larger lesions, in death. During survival, such comatose animals show persistent slow waves in their EEG and cannot be aroused by sensory stimulation. The potentials appearing in this system after sensory stimulation are greatly reduced and eventually abolished when the animal is anesthetized or the oxygen and sugar supply of the brain are lowered. At the same time, the sharp spike potential appearing in the sensory cortex on arrival of sensory impulses is not diminished, and may even be augmented. Conversely, strychnine in subconvulsive dosage markedly increases the activity in this "activating system" but does not change the sensory potentials.

This system is generally believed to be fed by collaterals from sensory pathways reaching from brain stem to thalamus. Its thalamic portions seem to be identical with the diffuse thalamic system, though it has been found that the cortex can be activated (as shown by EEG arousal) even when the thalamus is destroyed (Starzl *et al.,* 1951). We venture to suggest, however, that the brain stem reticular formation with its connections to the diffuse thalamic system represents a series of relays in a system mediating the appraisal of sense impressions, which we have called the estimative system. We propose to show that this system receives peripheral afferents and has a specific projection to the cingulate gyrus and other limbic cortical regions, and a diffuse projection (via the reticular thalamic nuclei) to the whole cortex.

The peripheral estimative system. In a brilliant exposition, Bishop (1959) has shown that there are five somesthetic pathways from peripheral receptors to the thalamus; but three of them go directly or indirectly (via relays in the brain stem) to the intralaminar nuclei, and only two (the medial lemniscus and the neospinal tract) go to the ventral thalamic nuclei (the somesthetic relay stations) and project to the somesthetic cortex. (The intralaminar nuclei are not part of the sensory system but of the estimative system.)

There is a difference in fiber size between the tracts that are part of the sensory system and those that connect with the medial thalamus and are, we think, part of the estimative system. The larger the fiber, the faster it conducts and the lower its threshold for excitation. In any given nerve, there are bundles of larger and smaller fibers which can be distinguished by the size and latency of the electrical potentials they produce on stimulation. The first and largest potential is recorded from the group containing the largest fibers, the following potentials from the bundles of smaller fibers. Three types of nerves have been distinguished according to the form of the potential waves they produce: nerves with A waves are medullated, rapidly conducting, and contain alpha, beta, gamma, and delta fibers; B wave nerves are considerably slower and have finely medullated fibers; and C wave nerves are unmyelinated and have the slowest conduction time.

The four fiber components of the A wave nerves conduct at different rates, according to their diameter. Alpha fibers are the largest and conduct at 100 meters per second; beta fibers, next in size, conduct at 60 meters per second, gamma fibers at 40 meters, and delta fibers at 25 meters per second.* Alpha fibers come from muscles and joints and run in the dorsal spinocerebellar tract to the cerebellum. Impulses conducted over these fibers are not experienced as sensations. Beta fibers (and only these) run in the medial lemniscus to the somesthetic relay station in the ventral thalamus and to the somesthetic cortex. They mediate touch, vibration, and sensations from muscles and joints. The fibers in the gamma and delta range run in the spinothalamic tract to the centrum medianum and ventroposterolateral nucleus in the thalamus, where they join the somesthetic projection to the cortex. They mediate sharp, adequately localized pain (Bowsher, 1957). We suggest that these fibers belong to the estimative system, and project to the posterior cingulate gyrus and the posterior insula.

Apparently, fibers from the same sensory (A) nerve divide and send one contingent (alpha fibers) to the cerebellum, another (beta) to the somesthetic cortex, and a third (gamma and delta) to the medial thalamus and limbic cortex. The relay to the cerebellum seems to mediate impulses to reflex adjustment of posture (cutaneous nerves have no alpha fibers); the relay to the somesthetic thalamic nuclei mediates the experience of the *quality* of sensation (touch, etc.); on arrival in the cortex, the exact locus of sensation in the body is experienced as well. In contrast, the relays to the medial thalamus seem to mediate the *effect* of stimulation (painful or pleasurable), and those reaching the limbic cortex, the *appraisal* of its meaning for the individual.

Nerves with B wave potentials conduct at a considerably slower rate (10 meters per second) than any of the components of the A type nerves;

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* The alpha, beta, gamma, and delta fibers producing the A wave have no connection with EEG rhythms.

and C type nerves which have the smallest diameter and are unmyelinated conduct at the rate of 2 meters per second. Both B and C type nerves mediate pain, tickle, itch, cold, and warmth. But these nerves, unlike the gamma and delta fibers of A type nerves, mediate diffuse pain, felt some time after the first, sharply localized pain. That the fibers in the so-called "pain and temperature" tract mediate pleasure as well as pain is shown by the fact that anterolateral chordotomy (which interrupts both spinothalamic and spinoreticular fibers) abolishes not only sharp and diffuse pain but also pleasurable sensations from the sex organs (Stookey, 1943).

Not only somesthetic but optic, olfactory, and auditory fibers also seem to connect with the estimative system. According to Bishop (1959), the largest fibers of the optic nerve relay through the lateral geniculate nucleus to the visual cortex (comparable to somatosensory beta fibers running in the medial lemniscus to thalamus and somesthetic cortex); the next smaller fibers, homologous with the gamma and delta fibers relaying to medial thalamus and limbic cortex, project to the lateral nucleus of the thalamus, which is connected with the medial thalamic region. And the smallest fibers, corresponding to unmyelinated somesthetic C fibers, relay to the tectum. It follows that the visual system, like the somesthetic system, seems to have peripheral fibers belonging to the estimative system. The sub- (or pre-) thalamic connection of this system with the action circuit seems to make it possible for man or animal to avoid harm by reflex movements (e.g., the scratch, pupillary and blink reflex, startle, etc.), while the thalamic connection seems to mediate actions that need only immediate, automatic appraisal (e.g., shielding one's face from a blow; the rage of the decorticated animal). The cortical portion of the estimative system, finally, seems to mediate the appraisal for appropriate action (see Fig. 10).

This system can account for all the findings that have led to the conception of a "brain stem activating system." Among these findings, for instance, is the report of Starzl and coworkers (1951) that electrical potentials appear in the ventromedial thalamus and midbrain tegmentum when the sciatic nerve is stimulated, even after the sensory pathway (medial lemniscus) is destroyed at the intercollicular level on both sides, and no potentials can be found in the somatosensory cortex. This may mean, as the authors suggest, that the connection from the sensory to the activating system is made by collaterals below the lesion. But it is also consistent with our view that the stimulus has excited sensory afferents relayed in the spinal lemniscus system and also finer fibers relayed in the anterolateral tracts to the intralaminar nuclei (see Bishop, 1959).

Activating system and action circuit. Starzl and his associates found that

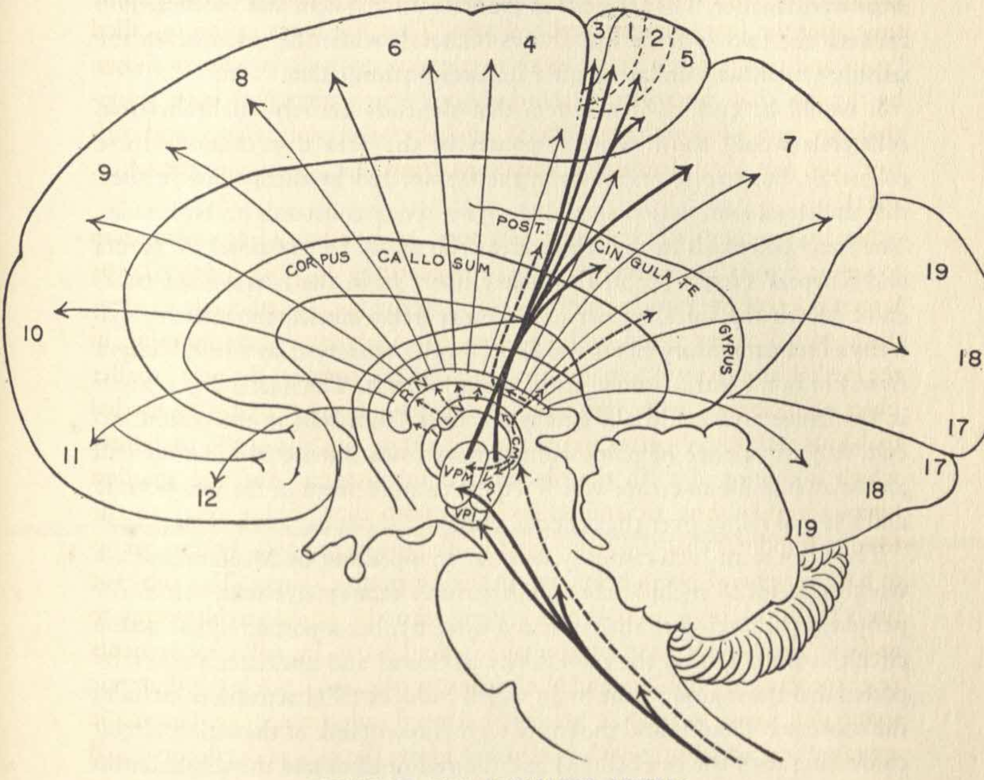

FIGURE 10. THE ESTIMATIVE SYSTEM

Peripheral fibers project in spinothalamic and spinoreticular tracts to intralaminar nuclei and posterior cingulate gyrus. The associated somesthetic projection is also shown. (Estimative fibers from other sensory systems are not sketched.) The diffuse projection from the intralaminar thalamic nuclei reaches every area of the cortex via the thalamic reticular nuclei.

▬▬▬ Sensory projection. - - - - - Projection of estimative system. ——— Diffuse projection. Arabic numerals refer to Brodmann areas.

CM centrum medianum. ILN intralaminar nucleus. RN reticular nucleus. VPI ventropostero-inferior nucleus. VPL ventroposterolateral nucleus. VPM ventroposteromedial nucleus.

Source: Outline of brain structures, from W. J. S. Krieg. 1957. Brain mechanisms in diachrome. 2d ed. Brain Books, Evanston, Ill. Pathways specially drawn.

the stimulation of sensory nerves induces potentials in the region between the sensory pathways and the brain stem reticular system, as well as in the regions that contain these systems, although the potentials in these fringe areas were smaller. The response in the activating system and in the region between the two systems was always bilateral, while the response in the sensory system was unilateral after unilateral stimulation.

It would be curious if a system that depends entirely on brain stem collaterals would show weak responses in the area that contains these collaterals, but strong responses in the system fed by them; and further, that unilateral stimulation should always activate collaterals on both sides. The small potentials in the fringe areas can easily be explained by Nauta and Kuypers' (1958) report that a few fibers from the lateral cord tracts end between the intralaminar and sensory relay nuclei, though the majority of somatosensory fibers end either in the sensory relay nuclei (larger fibers) or in the intralaminar nuclei (small fibers). The bilateral potentials in the fringe area could also be the response from cells in the action circuit. Any experience of pain or discomfort from intense stimulation will arouse an impulse to escape which requires a movement of the whole body and bilateral relays over the action circuit.

The brain stem "activating system," as mapped out by Magoun and coworkers, seems to include not only structures that receive relays from the periphery (belonging to the estimative system), but a portion of the action circuit as well. This is the more likely as Starzl and associates (1951) reported that the region in the brain which induces EEG activation includes the superior colliculus and the entire tegmentum (link of the action circuit connecting with the cerebellum) and the red nucleus and the subthalamus (part of the extrapyramidal pathway), but in addition the median center nucleus and reticular nucleus (in the diffuse thalamic system), the ventromedial and ventrolateral thalamic nuclei (which receive the projection from the cerebellum), and the area next to the somatosensory relay nuclei. That this EEG activation is the result of an impulse to action carried over the action circuit seems supported by the reported latencies of sensory stimulation. For instance, the electrical potentials in the sensory pathway (medial lemniscus) occurred 6 to 9 milliseconds after sciatic stimulation, and in the somatosensory cortex, after a delay of 10 to 12 milliseconds. But in the tegmentum they appeared after 13 to 23 milliseconds, a delay that could not be accounted for by a relay over short collaterals in the brain stem. Similarly, Collins and O'Leary (1954) recorded potentials in the pretectum after a brief electric shock to the contralateral radial, saphenous, and sciatic nerves. After comparing the excitability cycles in the parts of the sensory pathway and pretectal areas from which they recorded, these workers decided that it was unlikely that axons or collaterals of the brain stem sensory tracts could have produced these potentials. Here, as in the findings of Starzl and associates, the electrical potentials in the "activating system" seem to have been produced by relays over the estimative system and action circuit. Electrical stimulation of a sensory nerve produces discomfort if not pain, and the normal reaction is to look toward the sensitive spot or to escape from such stimulation.

Even if no action is taken, sensory stimulation will induce motor impulses that prepare for action. This has been confirmed by experimental evidence. For instance, Wall *et al.* (1953) found that electrical stimulation of the motor cortex was facilitated by various types of sensory stimuli. A single light flash was sometimes sufficient to induce a volley of motor impulses in the pyramidal tract. Sensory stimulation always intensified the electrical activity produced in this tract by stimulating the motor cortex. This effect disappeared after making a deep cut through the side of the brain stem just in front of the superior colliculus, a cut which apparently destroyed a direct pathway from the lateral geniculate nucleus (the relay station for visual impulses) to the tectum.

When a sensory experience is actively focused on, there seems to be an influx from the action circuit back to the sensory system, which may intensify the experience. This happens most strikingly in pain. Intense pain arouses a strong impulse to escape which cannot be denied by diverting attention away from it. We are forced to focus on the pain, thus increasing our discomfort. On the other hand, when we are engrossed in something that holds our attention, moderate pain seems to fade or, at least, is felt as less distressing. This may be the result of a lack of reinforcement of sensory impulses by collaterals from the action circuit. During relaxation, there may even be a reduction of somatosensory impulses, produced by the general inhibition of motor impulses via the action circuit. We have mentioned before that such an inhibition of proprioceptive, vestibulary, visual, and olfactory impulses can be obtained by stimulating the inhibitory regions in the brain stem reticular system (see Ch. 4). This increase or reduction of sensory impulses may conceivably be initiated via efferent fibers in the dorsal sensory roots which have been reported by many investigators (cf. Toennies, 1938; Lewis, 1942).

Waking and sleep after brain stem transections. We have suggested that the spontaneous recall necessary for identifying something and appraising it is mediated by a circuit that includes the hippocampus, brain stem, and sensory nuclei, while the exercise of imagination is mediated by a shorter circuit via the amygdala and the thalamic association nuclei. The urge to action, as we conceive it, requires a relay of neural impulses from the hippocampus via the brain stem and cerebellum to the frontal lobe. If our hypothesis is correct, there should be a considerable difference in EEG activity when the brain is isolated from the body in such a way that this action circuit is interrupted, as compared with a cut which leaves this circuit intact. This would also apply to an interruption of the memory circuit. When the brain stem is cut behind the pons, the action circuit and the memory circuit remain intact. This preparation is called an "encéphale isolé." When the brain stem is cut between the superior and inferior colliculi, or just behind the latter, the action circuit is interrupted; this preparation is a "cerveau isolé." We will now briefly review the differences that have been found in these two types of brain stem transections.

The *encéphale isolé* shows a normal sleeping and waking rhythm in the EEG, though the animal is paralyzed from the neck down. (From Dandy's case of a woman with a complete lesion of the cervical spinal cord, who was paralyzed and insensitive from the neck down, we know that such injury leaves intellectual functions, emotions, and general awareness unchanged.) When the encéphale isolé is asleep, the same stimuli will wake it that wake a normal animal. As in the normal animal, electrical stimulation of some cortical areas produces EEG arousal; so does injection of adrenaline, amphetamine, and lysergic acid (Bradley and Elkes, 1957).

The *cerveau isolé,* on the other hand, does not respond like a normal animal. When the brain stem is cut between superior and inferior colliculi, or just below the inferior colliculi, the animal is not only paralyzed but asleep, though olfactory stimuli can produce EEG arousal for a short time. Adrenaline and lysergic acid do not induce EEG activation, and amphetamine does so only when the mammillary bodies are included in the cerveau isolé (Bradley and Elkes, 1957).

When the midbrain is cut at the intercollicular level but the cerebral peduncles are spared and the connection between the motor cortex and spinal motor pathways is intact, the EEG shows the same permanent sleep pattern as in the cerveau isolé. Though the motor pathway is undisturbed, there is no spontaneous movement of ears or eyes, which shows that such movements need more than transcortical connections. Even the intact imagination circuit, which apparently is responsible for the short EEG arousal after olfactory stimuli, is not sufficient to induce movement or emotion. Waking consciousness, EEG arousal, emotion, and action seem to depend on the memory circuit and the action circuit interrupted in the cerveau isolé.

According to Rossi and Zirondoli (1955), cutting the brain stem rostral to the trigeminal nerve in the medulla oblongata also produces a "cerveau isolé" and blocks the EEG activation induced by inhalation of volatile anesthetics. When the brain stem is cut just behind the trigeminal nerve, however, EEG activation is undisturbed. Since pretrigeminal transection excludes the influx of impulses from the cerebellum, these authors removed the cerebellum by suction and reported that this "did not modify the effects on cortical rhythm of brain stem transections performed at different levels" (p. 385). Since impulses to and from the cerebellum synapse in the brain stem, this result is not unexpected. Blocking of the EEG activation by pretrigeminal transection seems to indicate that the nuclei at the caudal border of the pons are necessary for maintaining some connection between the hippocampus and the frontal lobe; hence, the action circuit may include the olivary nuclei. The fact that EEG activation was undisturbed after removal of the cerebellum does not prove that action impulses (including emotion) normally avoid the cerebellum; it merely demonstrates that the relay via the brain stem can produce EEG activation in the absence of the cerebellum. Impulses to action, no doubt, can be felt after the cerebellum is removed, but action itself is seriously disturbed.

It is usually assumed that the difference between the cerveau isolé animal (which is permanently asleep and shows brief EEG activation only on olfactory stimulation) and the encéphale isolé (which has a normal waking and sleeping rhythm) can be accounted for by the number of afferent impulses arriving in the lower portion of the ascending reticular system (cf. Bremer, 1954). But there are at least olfactory and visual impulses arriving in the sensory cortex of the cerveau isolé (Arduini and Moruzzi, 1953). They might be expected to keep the animal awake if the waking state really is the result of afferent impulses connecting with the reticular system throughout its length. At worst, the waking state might be reduced and sleep lengthened because there are fewer impulses demanding action. Similarly, a cut behind the trigeminal nerve should merely lengthen the time of waking because this nerve also can send collaterals to the reticular system.

In a later investigation, Roger, Rossi, and Zirondoli (1956) destroyed the Gasserian ganglion in an encéphale isolé and thus eliminated the trigeminal nerve at its source. They found that this procedure produced sleep spindles not only in the somatic area (analogous to the sleep spindles in the visual or auditory areas when optic or auditory nerves are destroyed) but also in the frontal area; hence, they suggested that the generalized sleep pattern of the cerveau isolé and the pretrigeminal transection stems from the lack of afferent trigeminal stimulation.

However, Batini *et al.* (1958) reported that a midpontine brain stem transection slightly rostral to the trigeminal roots, interrupting all connections between this nerve and the cortex, showed a waking EEG pattern with low voltage fast rhythms. But a transection through the rostral part of the pons (apparently comparable to Roger *et al.*'s "pretrigeminal transection") showed a permanent sleeping rhythm with high voltage slow activity. The midpontine section produced an animal which could follow a moving object with its eyes and could react to an emotional stimulus (dog or mouse) with pupillary dilatation. Hence, it seems clear that the elimination of the trigeminal roots does not eliminate the waking EEG pattern. In fact, these cats showed a fast waking pattern for 70 to 90 percent of the recording time, while normal intact cats, equally isolated in a quiet environment, usually show waking rhythms only for 20 to 50 percent of the time. We would suggest that the radical reduction of sleep, as shown by the EEG record after midpontine pretrigeminal transections, occurs because the connection with the cerebellum is broken. Without the amplification and organization of the impulse to relax which occurs in the cerebellum, cerebral activity apparently cannot be reduced sufficiently.

From the above survey of EEG studies after brain stem transections we may infer that waking EEG patterns and waking consciousness depend on optimal functioning of the estimative system, and on intact action and memory circuits. If the action circuit includes the cerebellum, there will be a regular alternation of sleeping and waking. If the cerebellar influence is eliminated, muscular relaxation apparently cannot become general, and sensory as well as imaginative activity cannot be depressed sufficiently; hence sleep will be short and sporadic. When the action and memory circuits are interrupted, psychological activity cannot be maintained for any length of time, and directed activity becomes impossible. Imaginative activity continues for a short time after stimulation, which accounts, for instance, for the short period of fast electrical activity in the cerveau isolé after olfactory stimulation.

THE SIGNIFICANCE OF THE EEG

Jung (1953) has pointed out that relays from a projection system, whether specific or diffuse, are not enough to account for the widespread electrical changes at the cortical surface that are reflected in the EEG. Rather, these wave patterns seem to be produced both by projection activity and by slower processes of excitation within the cortical feltwork.

We have argued that the recruiting response which is projected via the diffuse system to the cortex is initiated via the hippocampus and the action circuit, with relays to the reticular thalamic nucleus and the caudate nucleus; and we have seen that the latter has both facilitatory and inhibitory effects. Now we suggest that this diffuse projection serves either to excite cortical areas or to depress them, depending on the rate of conduction. Hence the fast activity called "EEG arousal" would be a sign that the activity in progress is facilitated by the simultaneous excitation of various cortical areas, while the slower alpha rhythm during rest would signify that this activity needs only moderate facilitation. When the rhythms are slowed further, first to sleep spindles and finally to the slow waves of deep sleep, the excitability of the cortex is reduced more and more, apparently through the recruitment of more and more units via the action circuit and the caudate nucleus.

Alertness and unconcern. The facilitatory influence of the caudate nucleus upon the diffuse projection to the cortex may represent the increased alertness implied in identifying a situation, recalling relevant memories, and imagining what is to happen and what to be done. Such a preoccupation with various aspects of the situation, which may allow accurate appraisal, is something that comes with increased experience. During infancy and early childhood, the child responds to the first aspect of the situation he grasps. He sees something and reacts to it wholeheartedly, but turns to something else just as promptly and easily. Gradually, the child learns to consider several aspects of a situation one after the other, and so is able to prolong his attention by reducing the appeal of other things.

This alertness to one thing and unconcern for others seems to be made possible by the relay from the hippocampus to the caudate nucleus and the diffuse thalamic system. When man or animal is preoccupied with something, the imagination and memory circuits are active and send relays via the action circuit to the caudate nucleus which facilitates action. This relay to the caudate and the diffuse thalamic system would account for the appearance of fast frequency waves in the EEG, the "arousal" response. Alpha waves seem to indicate the unconcern typical for rest. Memory and imagination are active but not directed toward anything in particular. Thus we have the persistent alpha waves in the occipital areas of people who use motor and auditory images rather than visual imagination (cf. Walter, 1953).*

Our hypothesis would explain why the EEG records of young children show delta and theta waves but no alpha rhythms. Even when these begin to appear (around three or four years of age), the EEG still shows mingled theta and alpha waves for some years, at least until about ten years of age, when it begins to resemble the adult pattern (Walter, 1953). The delta waves in early childhood and the later theta waves seem to indicate impulsive reactions, with no attempt to appraise the situation accurately, and hence little imaginative and memory activity. This seems to be the reason why children, adolescents with behavior disorders, and aggressive psychopaths show prominent theta rhythms. It also seems to explain why theta rhythms appear when some pleasure is withdrawn, or during laughter or weeping (Walter, 1950); during pleasant stimulation, imagination is active, but when it stops, there is a momentary gap in mental activity until something else attracts the attention. During laughing and crying, there is little imaginative or memory activity. Walter also mentioned that visual stimulation at the frequency of the theta rhythm is unpleasant, even annoying; but if the person is encouraged to control his annoyance, the theta rhythm eventually gives way to normal faster waves. Apparently, the thinking necessary to control his feelings makes it possible for a man to dampen cortical excitation and so to tolerate the stimulation. While children have too little concern for the various aspects of a situation, neurotics have too much. They ceaselessly worry about what might happen and how to guard themselves from every possible danger. Hence, their EEG shows predominantly fast frequencies.

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* Darrow's observation that stimulation and EEG activation are followed by increased anterior dominance of alpha-like activity in motor and frontal areas of emotionally unstable and brain-injured adults seems to indicate that these people cannot formulate a plan for action immediately, and thus the motor and motor association areas remain at rest. In contrast, well-adjusted adults show posterior alpha dominance under the same circumstances. They immediately imagine what is to be done; this activates the frontal lobes while the postcentral sensory areas are quiescent and show increased alpha activity (see our discussion of Darrow's theory earlier in this chapter).

Drug effects. Studies on the effect of various drugs on the EEG have provided evidence that is consistent with our hypothesis. For instance, the intracarotid injection of *atropine* (which prevents synaptic transmission in cholinergic nerves) abolishes the arousal effect and produces a sleep pattern in the EEG, although the individual is wide awake and may even be excited (Bradley and Elkes, 1957). Conversely, *acetylcholine* (the synaptic mediator in cholinergic nerves) and anticholinesterase (which preserves this mediator) produce EEG arousal in the resting animal. When the *corona radiata* is cut, this arousal is *prevented* on that side even if the blood supply is intact; hence, fast frequency waves seem to be conducted to the cortex over the diffuse projection from the thalamus. After an injection of atropine, sensory stimulation does not induce arousal, but electrical stimulation of the medial tegmentum does (Rinaldi and Himwich, 1955b), which indicates that the projection mediating arousal is cholinergic, but that intense stimulation can break through the blocking action of atropine. A blocking of the diffuse projection to the cortex would not affect the action circuit, but would prevent the increase of alertness by imagining and remembering and might make man or animal more distractible. Consequently, atropine does not immobilize the animal and does not prevent excitement.

Since the EEG arousal by acetylcholine is not prevented by cutting the spinal cord or lower brain stem (in the encéphale isolé), it cannot be mediated by relays from the periphery. Indeed, the drug has the same effect on the EEG of the cerveau isolé with a high midbrain transection which excludes the posterior hypothalamus (Rinaldi and Himwich, 1955b). Substances injected into the blood stream cannot penetrate the blood-brain barrier but can invade the hypothalamus, probably via the pituitary portal system (Leonhardt, 1955). Thus any drug can affect the thalamus and cortex only by affecting hypothalamic cells that project to these areas. Since acetylcholine induces EEG arousal and atropine prevents it, these relays to the cortex must be cholinergic.

Intravenous *adrenaline* does not produce EEG arousal in the cerveau isolé (even when the mammillary bodies are included with it), but does induce it in the encéphale isolé. Apparently, adrenaline cannot affect the EEG via direct connections from the hypothalamic receptor cells to the diffuse thalamic projection because these, as we have seen, seem to require a cholinergic mediator. We know, however, that the hypothalamus contains adrenaline- and noradrenaline-sensitive fibers. Vogt (1954) has reported that the noradrenaline content of the hypothalamus is five times that of the medial thalamus. Sympathin, the synaptic mediator in the sympathetic nervous system, which contains both adrenaline and noradrenaline, may mediate the transmission of nervous impulses in the hypothalamic branch of the action circuit by exciting these sensitive fibers. Relays to the prefrontal areas would be felt as impulses to action and appraised via the anterior cingulate gyrus which connects with the hippocampus and, via the action circuit, with the diffuse thalamic projection to the neocortex. In the cerveau isolé, adrenaline probably affects the relays to the prefrontal cortex but cannot affect the EEG because the connection from the action circuit via the cerebellum and midbrain to the medial thalamus has been interrupted by the cut.

Adrenaline and related compounds reduce and eventually inhibit the synaptic transmission in the brain, the spinal cord, and autonomic ganglia (Marrazzi and Hart, 1957). Noradrenaline requires fifteen times the dosage to produce the same effect. This means that all psychological processes will be retarded when the adrenaline secretion is substantially increased, as, for instance, in fear. Recall, imagination, and even the impulse to action will be equally affected. This may account for the fact that intense chronic fear may immobilize animals (Ch. 7) and that even acute fear may result in a complete inability to remember (e.g., in stage fright) or to act (e.g., the novice pilot "freezing to the controls"). Noradrenaline, which seems to be secreted during anger, does not usually have this effect; but excessive secretion will also retard psychological processes. Thus a man may become inarticulate when he is very angry.

Amphetamine is related to adrenaline but does not excite the sympathetic nervous system and does not seem to inhibit cerebral synapses. Unlike adrenaline, it does induce EEG activation in the cerveau isolé, provided the mammillary bodies are included in it (Dell *et al.,* 1954). Hence, amphetamine seems to stimulate fibers that project from the mammillary bodies to the anterior thalamic nuclei which are connected both with the diffuse system and with the cingulate gyrus. We have suggested before that the connection from the mammillary bodies to the anterior thalamic nuclei and the cingulate gyrus of both sides serves the recall of success and failure, and that the anterior cingulate gyrus mediates the appraisal of actions and action impulses as suitable or unsuitable. The stimulation of this structure by amphetamine would account for the feeling of alertness and the general readiness for action felt after taking this drug; it would also account for the fact that amphetamine does not affect the EEG in the cerveau isolé when the brain stem is cut in front of the mammillary bodies. If amphetamine only excites the cingulate gyrus and the thalamic estimative system, it is not surprising that the sympathetic stimulation (which is so prominent after adrenaline) should be absent; for autonomic motor nerves are excited via the action circuit (see Ch. 3).

D-lysergic acid (LSD-25) also alerts the EEG (Bradley, 1953), but its effect seems to depend on sensory stimulation, for it is abolished when the animal is resting in a quiet room or when most of the sensory nerves are excluded (in the encéphale isolé or the cerveau isolé; see Bradley and Elkes, 1957). Lysergic acid reduces and in larger doses blocks synaptic transmission in all layers of the lateral geniculate nuclei (Bishop *et al.*, 1958), which means that visual recall is probably difficult long before vision itself is affected, for the smaller fibers in these nuclei would seem to belong to the memory circuit and estimative system while the larger fibers seem to mediate visual sensations; and small lightly myelinated fibers are more susceptible to drugs than larger fibers. Other sensory relay nuclei and synapses seem to be depressed also (Marrazzi and Hart, 1957). Hence sensations will be received but cannot be appraised accurately; nor can they be identified easily, which accounts for the feelings of strangeness reported after LSD-25 injection. In addition, sense experiences will arouse abundant fantasies, seeing that the memory circuit is depressed; this probably accounts for the reported hallucinations. The increased imaginative activity, reduced recall, and difficult appraisal would make for the distortion of perception that is so characteristic for the drug.

At the same time, lysergic acid seems to have no positive effect of its own, only the negative one of depressing sensory synapses; for it does not affect either the cerveau isolé or the encéphale isolé. It cannot affect the diffuse system via the action circuit, for if it did, the encéphale isolé would show the arousal changes in the EEG, as it does, for instance, after adrenaline. Apparently, the effect of lysergic acid depends entirely on the reception of sensory impulses which the individual will experience in a thoroughly abnormal way. This very difference in experiencing his usual surroundings and his own body will arouse apprehension—which will initiate action impulses with relays to the diffuse thalamic system. In contrast, the resting encéphale isolé, with eyes closed, receives only olfactory impulses. Even though these arouse imagination without the usual recall, the difference is not alarming enough to arouse fear. Hence the drug does not affect the EEG in the encéphale isolé but does do so in the intact animal.

Various *tranquilizers* also affect brain function. They seem to have a double effect and either of the two may predominate with a given drug: they seem to block hallucinations and they also induce relaxation (Barsa, 1957). They also seem to induce increased clarity of thought, better memory, and greater ease of report (e.g., on the effects of Frenquel, see Toll, 1957).

Frenquel, reserpine, and chlorpromazine improve the conduction in sensory cerebral synapses and antagonize the inhibitory effect of LSD, mescaline, and adrenaline on these structures (Rinaldi and Himwich, 1955c). They also block EEG activation by amphetamine and LSD-25 (Marrazzi and Hart, 1957; Bradley and Hance, 1957). These findings suggest that tranquilizers facilitate the memory circuit, improve recall and appraisal, and so normalize imagination. The reported "volitional reduction," as well as the lessening of tension and anxiety (Sainz, 1956), may well be the result of this shift in mental activity. When memory dominates imagination, there will be little apprehension. Everything will be identified and appraised according to its past effects and there will be no room for doubt as to its future potentialities. With recall dominant over imagination, there will be few impulses to action, for it is what we expect to happen that decides us to do something about it. This reduction of action impulses accounts for the reduction of tension, but it also accounts for the lack of enthusiasm and initiative, even the depression, that is so often reported in long term treatment. The impoverishment of imagination may also account for the hormonal deficiencies that may appear after some time. Reserpine, for instance, produces castration-like changes in the genitals of rats (Tuchmann-Duplessis, 1956); and impotence with reduction of sexual desire is a common side effect of reserpine and chlorpromazine treatment in man. That this may be the result of a depression of imaginative activity is suggested by the finding that destruction of the amygdala (which initiates, we think, relays over the imagination circuit) results in atrophy of the endocrine glands in puppies (Koikegami *et al.*, 1955). Such results would suggest that reserpine inhibits synaptic transmission in the amygdala; a sample record from Monroe *et al.* (1955) would indeed confirm this notion.

Both barbiturates and reserpine have been found to depress hippocampal activity (MacLean *et al.*, 1955/56), barbiturates because they depress cortical electrical activity and some of them also the activity of the caudate nucleus and the thalamus (Monroe *et al.*, 1955), and reserpine perhaps because it depresses the amygdalae, so reducing imagination. This would explain why volitional activity becomes increasingly difficult with long reserpine (or chlorpromazine) treatment, to the point where the patient is unable to carry out his usual routine unless he is told what to do (Sainz, 1956).

Meprobamate (Miltown), another of the tranquilizing drugs, seems to have a powerful relaxing effect but does not seem to block hallucinations (Barsa, 1957). According to Hendley *et al.* (1957), it synchronizes the thalamic rhythm at 10 to 15 cps. (in the cat) but does not affect the cortical rhythm. These authors assume that this implies a slowing of thalamic activity; but Rinaldi and Himwich (1955a) have found that arousal accelerates and regulates the rabbit's thalamic sleep pattern (mingled 2–3 cps. and 5–6 cps. waves) so that it becomes a 6 cps. arousal pattern, which would suggest that meprobamate actually accelerates the waking rhythm. This would mean that alertness is increased by faster conduction in the diffuse system and would account for the fact that meprobamate sometimes induces sleeplessness, though usually the patient merely wakes easily and is immediately in contact with the environment. As with Frenquel, thinking becomes easier and rapport is improved. In addition, meprobamate seems to have a more pronounced relaxing effect than other tranquilizers. This might be the result of the increased efficiency of the estimative system, indicated by the faster thalamic rhythm. If everything can be appraised immediately and correctly, there is no reason for uncertainty or apprehension. Since imagination is not blocked, action will become quick and assured, and tension will decrease.

The whole field of psychopharmacology is only at its beginning. The interpretation given here is admittedly tentative, even speculative. At the same time, it is consistent, agrees with the evidence, and rests on a coherent theory of brain function. Perhaps it is not too much to hope that this theory will make it possible to test drug effects on psychological functions in a more exact way.

6. PATTERNS OF ACTION

In the last four chapters we have marshalled evidence which seems to indicate that every action is mediated by a relay of neural impulses from sensory and association areas to the limbic cortex and the hippocampus, and from there to midbrain, cerebellum, thalamus/hypothalamus, and the frontal lobe. We have also reviewed evidence which seems to show that an impulse to action is often, if not always, accompanied by some autonomic changes and even by some stimulation of the endocrine system.

There are various patterns of action: there are actions rooted in a hormonal change that leads to seeking a particular kind of object appraised as good, thus arousing instinctual desire and culminating in instinctual satisfaction. Among these *instinctive action patterns* we shall discuss hunger and eating, sexual desire, and mating. Next, there are *emotional action patterns* which take their origin in the appraisal of something perceived or imagined as beneficial or harmful, arousing various emotions and leading to appropriate actions. Among them, we have most information on fear and anger, and shall confine our discussion to these. There are also action patterns that are initiated on the basis of a *feeling* rather than an emotion, of a reaction to something appraised as beneficial or harmful *for our functioning*. Since some experiences felt as unpleasant have profound effects on the body, the pattern of action designed to avoid them includes equally strong countermeasures. We propose to discuss three such action patterns, those connected with the avoidance of heat, cold, and pain. By mapping out these various action patterns in detail, we hope to lay a foundation for our discussion of the peripheral effects of emotions and feelings in the next chapter.

INSTINCTIVE PATTERNS: I. HUNGER AND EATING

According to our hypothesis, eating is initiated by an appraisal of something as good to approach, in particular, by the appraisal that this is good to eat and the intention to approach it, take it, and finally eat it. When we decide to eat because we see a tempting morsel or because dinner is set before us, the neural circuit mediating this action presents no particular problem. There is a wanting, which we might call *appetite,* and this tendency urges to an action that is, as usual, mediated by relays from hippocampus to cerebellum and frontal lobe.

Hunger and food seeking. When we neither see something that tempts our appetite nor are reminded of anything we want to eat, yet feel hunger, the matter is more complicated. Hunger includes not only a felt organic sensation but also an urge to think about food (imagine it) and, in addition, to look or plan for it. "Hunger pangs," so-called, are part of the feeling of hunger. They are contractions of the stomach and duodenum (Carlson, 1916; Quigley, 1955) which are felt as organic sensations. They can be increased by stimulation of the vagus nerve, by fasting, anger, moderate exercise, exposure to cold, and injection of insulin. Of the conditions that increase hunger pangs, some are certainly induced by central nervous excitation (for instance, anger), while others seem to depend on metabolic changes induced by an increased need for food. Hunger contractions occur even in the denervated stomach, which seems to suggest that metabolic changes alone can produce them, though normally central nervous excitation seems to be involved as well (Quigley, 1955).

This excitation seems to be relayed from the hypothalamus. Several investigators (see Larsson, 1954; Miner, 1955) have found that electrical stimulation of the lateral hypothalamic nucleus, including a strip to the optic chiasm and beyond, induces prolonged eating; and Forssberg and Larsson (1954) have reported that fasting is accompanied by increased uptake of glucose and phosphorus in the lateral hypothalamic nuclei. These workers suggested that the increased metabolic activity in these nuclei might constitute the driving force of hunger. The exact mechanism by which such increased activity in the lateral hypothalamus occurs has not been established; but we do know that the hypothalamus can detect and respond to a number of chemical stimuli. Accordingly, there may be receptors in the hypothalamus which register the level of some blood constituent that indicates tissue needs (for food, water, salt, etc.); and the receptors registering a depletion of this factor may activate relays to the lateral hypothalamus and so induce the increased uptake of glucose and phosphorus found by Forssberg and Larsson. Whatever the nature of this blood constituent turns out to be, it would seem that it stimulates the lateral hypothalamus during hunger and does not stimulate it during satiation.

If the lateral hypothalamic nuclei should relay this excitation to other areas, such relays might account for the various experiences connected with hunger. First of all, they should account for the gastric hunger contractions and the sensation of hunger. Now we know that there is a projection from the lateral hypothalamus to the frontal tip and caudate nucleus (Tankard and Harman, 1955) which connects with the globus pallidus, red nucleus, and vagus motor nerve. This projection could mediate stomach contractions which are felt and appraised as unpleasant. The caudate nucleus is also connected with the premotor and motor cortex, mediating an impulse to action which can impart urgency to the experience of hunger. Hunger, like emotion, seems to be a tendency to action based on the appraisal of something as unpleasant. But unpleasantness as such could only give rise to a decision to escape from it, as does, for instance, the extreme unpleasantness we call pain. Instead, hunger seems to urge us *toward food.*

It has often been supposed that the impulse to action aroused by hunger is simply restlessness, an increase in random activity; but that is an inference from laboratory experience. Only caged animals show increased random activity, simply because they cannot find food until it is brought to them. Animals living in their natural habitat do not pace planlessly; they begin to look for a place to graze or dig worms, or for a place to hunt. Such directed activity is possible only if man or animal is able to remember where food can be found and to imagine what to do to obtain it. Only when nothing can be done (because the animal is caged or domesticated), or when remembering and imagining are impossible (when cortex, hippocampus, or amygdala has been removed), will the impulse to eat be expressed as planless activity. Freud postulated long ago that every instinctive impulse arouses a fantasy of the object that can satisfy it (the so-called "primary process").

Immediately on feeling hungry, there seems to be an image or fantasy of the needed food. This image seems to be mediated by a relay from the lateral hypothalamus to the amygdala and various cortical association areas. A direct one-way connection from the lateral hypothalamic nuclei to the tip of the temporal lobe and the amygdala has actually been found (Stoll *et al.*, 1951); and the connection from amygdala to the association areas has been reviewed before (Ch. 3). The imagined food is next appraised as good to eat. This arouses an impulse to look for such food, and also seems to initiate the secretion of saliva and gastric juice. French and associates (1953) found that stimulation of the anterior hypothalamus yields early gastric hydrochloric acid secretion (mediated by the vagus motor nerve) which is at a peak an hour after stimulation. Electrical stimulation of the posterior hypothalamus induces a late hydrochloric acid secretion which comes to a peak in three hours and is mediated by the anterior pituitary gland and adrenal cortex. It would seem that the early secretion is the result of the impulse to look for food, mediated via the action circuit which is connected with the globus pallidus, premotor area, and bulbar motor nucleus of the vagus via both pyramidal and extrapyramidal pathways (see Ch. 3). The late secretion of gastric juice seems to be the result of a stimulation of the anterior pituitary gland via the action circuit which results in the discharge of adrenocorticotrophic hormones (ACTH) and the secretion of adrenal cortical hormones.

That appetite (the desire to eat) accompanies gastric hydrochloric acid secretion is shown by the fact that cancer patients have normal appetite as long as hydrochloric acid is found in the stomach, but lose their appetite when hydrochloric acid is no longer secreted (Gilmour, 1958). Our suggestion that the action circuit is activated when food is imagined or expected also explains why the stimulation of areas 6 and 8 produces salivation and secretion of gastric juice (Fulton, 1951). We have suggested that these cortical areas mediate the felt impulse to look for something or seek something; hence also, to look for food or hunt for it. Pavlov's conditioned salivary reflex seems to depend on the same mechanism, as does the often reported fact that a hypnotized person who imagines eating a particular food secretes bile and gastric juice in the same amount and of the same quality as he does when he really eats that food (cf. Barber, 1958).

A pathway between two nuclear masses, the lateral hypothalamic nucleus and the amygdala, can hardly allow for anything but an innate connection, probably resulting in an image of the naturally appropriate food. To allow for learning (e.g., recalling and visualizing foodstuffs that have proved palatable), we would expect a pathway with cortical connections. This is provided by the connection from the lateral hypothalamus to the temporal tip via the amygdala.

Felt hunger and the desire to eat. Hunger pangs, induced by connections from the lateral hypothalamus to the vagus nerve, we believe, are experienced in turn, an experience mediated by the somesthetic cortex. These sensations are unpleasant and disliked. The feeling of hunger, experienced as a gnawing discomfort, together with the imagination of some fancied food, will induce an action impulse which will be relayed from the hippocampus to midbrain, cerebellum, and, via the hypothalamus/thalamus, to the frontal lobe. The hypothalamic part of this circuit seems to join with the relays from the lateral hypothalamus, reinforcing the impulse to action which has now a definite direction.

That such a pathway exists and functions in hunger can be inferred from several research findings. Collins (1954), for instance, reported that monkeys with a small lesion along a tract connecting the midbrain tegmentum with the caudal hypothalamus and the lateral hypothalamic area, refused food and drink, lost weight rapidly, and showed a fall in heart rate, respiratory rate, and rectal temperature. These monkeys hardly moved, did not chatter, were drowsy and apathetic, and showed no facial expression for about two weeks after the operation. During this time, they easily succumbed to infection, ate when spoon-fed, but would not take food for themselves. After two weeks, they gradually improved, but temperature, heart, and respiratory rate never returned to normal. According to Collins, the tract interrupted by the lesion corresponds to the posterior portion of Crosby and Woodburne's hypothalamotegmental tract.* Lesions within this tract did not abolish eating (as did, for instance, septal area lesions, according to Heath *et al.*, 1954), but did abolish food seeking. Since the connections from the lateral hypothalamic nuclei to the frontal lobe and premotor area were intact, the animals may have experienced hunger but would not take food by themselves because nothing they saw aroused any desire or appetite. As they recovered, they seemed to have learned to let hunger alone guide them, without depending too much on the desire aroused by seeing and appraising food.

Stimulation of a strip from the lateral hypothalamus to the septum (evidently identical with Crosby and Woodburne's tract) produced chewing and licking, and increased eating. Since stimulation of this tract increases the intake of food which the animal likes and usually takes, rather than producing automatic chewing and swallowing of anything offered, this tract seems to connect with the premotor and motor cortex, as we suggest, and thus makes directed action possible. If this tract were a descending tract from hypothalamus to midbrain and spinal cord, as is usually assumed, stimulation should induce automatic chewing and swallowing rather than directed behavior. Stimulation of the medulla oblongata in an area near the dorsal motor nucleus of the vagus nerve, is actually followed by indiscriminate eating of everything, edible or not, as long as it can be chewed (Larsson, 1954); it is likely that this area is within the final extrapyramidal motor pathway.

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* Crosby and Woodburne (1949) describe a tract which collects fibers from the medial preoptic and anterior hypothalamic area along the ventromedial angle of the medial forebrain bundle and is joined by a tract from the ventromedial hypothalamic nucleus. Both tracts connect with the anterior part of the deep mesencephalic nucleus and send fibers beyond (destination unknown). Crosby and Woodburne assume that these are descending tracts, but the direction of degeneration is difficult to determine; thus it is possible that we are dealing here with ascending fibers.

While the impulse to find food is originally mediated by relays from the lateral hypothalamus, it is intensified by the sensation of hunger, which is aroused by hunger pangs. Even without this sensation, the sight of food can arouse appetite, a desire to eat, and it can also arouse hunger pangs, but only when there is a need for food. Shortly before dinner, just seeing a juicy steak may induce hunger. After a meal, it does not, though we may be tempted to take a candy without being hungry at all. Accordingly, we would suppose that the tract from cerebellum to hypothalamus and frontal lobe, which mediates the impulse to eat something seen and found appetizing, runs concurrently with the fibers from the lateral hypothalamus to the ventral thalamus and caudate nucleus but does not synapse with these fibers. Only when the hypothalamic receptors registering the need for food have excited the lateral hypothalamic nucleus does there seem to be a relay of nerve impulses to the vagus nerve, inducing hunger pangs.

When this combined pathway is interrupted between the lateral hypothalamus and the caudate nucleus by a lesion in the septal area, both hunger and eating are abolished. Two or three days after the operation, cats refused to eat even when food was put into their mouth. In spite of tube feeding, they lost 30 to 50 percent of body weight; blood sugar and glycogen stores in liver and muscles disappeared; and the animals died within a short time (Heath *et al.,* 1954). Since the relay from the lateral hypothalamus to the frontal lobe and the caudate nucleus was interrupted, the animals could have felt neither an impulse to eat nor hunger pangs. Even when they could see and appraise food, they could not have felt much desire to take it, for the action circuit connecting with the extrapyramidal pathway for eating was broken.

Thirst, salt hunger, and other hungers. We have suggested that the hypothalamic receptors detect not only a tissue need for food (glucose) but also a lowering of other vital constituents in the blood. This seems to initiate increased activity and induce neural impulses which are relayed to various connected structures. A similar mechanism seems to arouse thirst, if we may judge from Andersson and McCann's (1955) results. These investigators stimulated a region between the descending fornix and the mammillothalamic tract which extended from the dorsal into the ventral hypothalamus. Such stimulation with implanted electrodes produced increased drinking in the goat, sometimes licking of salt and, when the stimulating electrode was at the lateral and caudal border of this area, chewing motions. Destruction of this area resulted in a refusal to drink. Stimulation of an area from the fornix to the anterior commissure (lateral to the paraventricular nucleus) also induced drinking, inhibited diuresis, and prevented milk ejection. This indicates a secretion of antidiuretic hormone (ADH) together with the impulse to drink. Apparently, another area in the hypothalamus, close to the area mediating eating, can initiate the impulse to drink, and stimulate the posterior pituitary gland, probably via projections to the premotor cortex and to the preoptic and paraventricular nuclei. At any rate, Kovacs *et al.* (1954) found that thirsty rats showed marked changes in these two nuclei as the result of increased secretion of ADH.

This mechanism seems to function also in salt hunger and may be active in other hungers as well. We know that animals select their diet so as to compensate for specific deficiencies. Richter (1954) and Young (in: Jones, 1955) have shown that such selection depends on the animal's ability to detect the needed food by taste or smell. According to Richter, salt hunger after adrenalectomy (and the resulting deficiency of mineralocorticoids; see Ch. 8) is accompanied by increased taste sensitivity for salt, so that adrenalectomized rats can detect one part of salt in 33,000 parts of water, as compared with normal rats who only detect one part of salt in 2,000 parts of water. Richter believes that this enhanced sensitivity is the result of chemical changes within the taste mechanism. Since there are connections from the hypothalamus to the olfactory nucleus, the septal and prepyriform area and the posterior insula (the areas responsible for smell and taste likes and dislikes; see Ch. 2), we can infer that the deficiency in a given blood constituent, detected by the hypothalamus, will alter the excitability of the limbic areas mediating the liking and disliking of foods that can supply what is lacking. In addition to making these foods more palatable, such increased activity of hypothalamic receptors will also induce an impulse to seek and eat the needed food.

Repletion and relaxation. When we are hungry, eating is pleasant. This pleasure is not merely the pleasure of smelling and tasting, but also an experience of repletion, of hunger stilled, which is as pleasant as hunger is unpleasant. The experience of repletion is based on various organic changes. Hunger pangs cease as soon as food containing sugar or fat or salt commences to enter the duodenum, apparently because a hormone, enterogastrone, is secreted there (Grossman, 1950). It is tempting to speculate that this hormone, or another one connected with it, may enter the blood stream, be detected by the hypothalamus, arrest the metabolic activity in the lateral hypothalamic nucleus, and increase this activity in the ventromedial hypothalamic nucleus. At any rate, the ventromedial nucleus is necessary for satiation. When it is destroyed, the animal begins to overeat and becomes obese (Miner, 1955). It has also been found that prolonged feeding through the jejunum combined with sham feeding by mouth brings on ravenous hunger in man and animals who then eat practically all day long (cf. Hollander *et al.,* 1955). In these cases, the impulse to eat apparently continues because there is nothing to inhibit the activity of the lateral hypothalamus. Forssberg and Larsson have shown that satiation is accompanied by decreased metabolic activity in the lateral hypothalamus and by increased activity in the medial and anterior part.

Though enterogastrone seems to act even in the denervated stomach, its usual action is via the vagus. In fact, enterogastrone extracts prepared thus far inhibit gastric contractions only when the vagus is intact. The relaxation of the stomach wall seems to be induced by a relay from the ventromedial hypothalamic nuclei via the septal area and caudate nucleus. From there, a relay to the premotor and motor region seems to mediate the relaxation of voluntary as well as involuntary muscles, while a relay to the globus pallidus, red nucleus, and motor nucleus of the vagus seems to mediate such relaxation via the extrapyramidal pathway. There is also a direct connection from the ventromedial hypothalamic nucleus to the globus pallidus, probably a part of the action circuit. This explains why arrest of gastric motility can be produced by cortical stimulation (see Fulton, 1949).

Vagal excitation can produce either gastric activity or gastric inhibition, probably depending on the rate of stimulation. We have seen before (cf. Ch. 4) that slow frequency stimulation in various brain structures induces relaxation while fast frequencies induce muscular contractions. Since the ventromedial hypothalamic nucleus as well as some nuclei in the anterior hypothalamus have a slow, 3 to 5 per second rhythm (Green and Morin, 1953), it seems possible that excitation of this nucleus by hypothalamic receptors induces slow frequency excitation, which is relayed to the caudate nucleus and globus pallidus and produces relaxation in both voluntary and involuntary muscles. We know that hunger pangs stop soon after eating begins and that the general muscular tension experienced in hunger begins to relax at the same time.

Excitation of the ventromedial hypothalamic nucleus seems to induce an impulse to imagine a place to rest and the act of relaxing (via a direct relay from this nucleus to the amygdala). This imagined arrest of action may be found suitable and carried out (via the hippocampal action circuit to the frontal lobe). After the first hunger is stilled, the thought may occur to a man to stop eating; but if the meal is appetizing or if he is too intent on the pleasure of taste, he may go on eating until the increasing distention of the stomach is felt as unpleasant. This feeling will arouse a renewed desire to stop eating, also mediated by the action circuit producing motor inhibition.

Finally, excitation of the hypothalamic ventromedial nuclei seems to be relayed via the caudate nucleus to the hypothalamic secretory nuclei * and the anterior pituitary gland, inducing the secretion of growth hormone or somatotrophic hormone (STH). This hormone stimulates the secretion of insulin and glucagon from the pancreas and so promotes the utilization of the food eaten (see Bullough, 1955).

Implications. The hypothesis stated above explains many recent findings. For instance, after destruction of the ventromedial hypothalamic nuclei rats overeat if the food is tasty but do not eat enough if the food is made more bulky by adding cellulose (Miner, 1955; Teitelbaum, 1955). They have lost the feeling of satiation and the impulse to stop eating but retain the pleasure of taste and so overeat. Food adulterated with cellulose is less tasty; hence animals stop eating it at the slightest discomfort from an extended stomach. Apparently, the changed composition of the food, probably resulting in less enterogastrone, can delay the impulse to stop eating in intact animals but has no such effect after loss of the ventromedial nuclei. Guided by taste alone, rats so affected refuse food to which quinine has been added, even when the bitter taste is so slight that intact animals show no distaste for it. Unable to experience satiation, they will not press a bar to obtain food for as long a time as normal animals do because food is not a reward for them but merely gives them an opportunity to do what their incessant hunger urges, namely, eat. Thus food does not motivate them in the way in which it motivates normal animals. If sugar were added to the food compound, making it more palatable, they might perform as well as normal rats.

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* The paraventricular and supraoptic nuclei are known to excrete posterior pituitary hormone, but opinions are divided as to whether they also secrete anterior pituitary hormones or whether these are secreted by other hypothalamic nuclei (see Harris, 1955; Ingram, 1956).

Miller *et al.* (1955, 1957) found that satiation is produced in normal animals when food is in the stomach, but is not felt when the stomach is merely distended. When rats in a simple T maze turned to the side chosen by the experimenter, they were given milk via a fistula so that it entered the stomach directly. Very soon, they learned to choose that side of the maze. Another group of rats had saline injected into a balloon within their stomach whenever they chose a particular side of the maze, and soon learned to avoid it. Thus milk in the stomach induced satiation; it was a reward. Sheer distention was uncomfortable; it acted as punishment. This explains why in another experiment both groups of animals soon ceased to work at a bar to get food: the milk-fed group because their hunger was stilled, the balloon-injected group because the discomfort in their stomach would not allow eating. Miller also found that milk injected directly into the stomach served as a reward in simple maze learning, but that milk taken by mouth proved to be a far stronger reward, as shown by faster learning. We have seen that eating brings not only the pleasure of repletion but also the pleasure of taste; thus learning will be faster if they are combined.

After destruction of the ventromedial hypothalamic nucleus or lesions of the ventral aspect of the septal area, animals become vicious and irritable (Wheatley, 1944; Spiegel *et al.,* 1940). In both cases, the relay from the ventromedial hypothalamic nucleus to the caudate nucleus seems to be prevented, so that the relaxation that accompanies repletion or other pleasures is no longer possible. The greatly increased activity resulting from destruction of the orbital cortex or from orbital undercutting (which interrupts a tract from the orbital cortex to the caudate nucleus—see Wall *et al.,* 1951) seems to be the result of a similar inability to relax. Animals so operated relax only during sleep, and sleep overtakes them suddenly.

Our hypothesis can also account for the intriguing results of septal area stimulation. Olds (1955, 1956) and Olds and Milner (1954) implanted electrodes in the septal area and other brain structures of rats and taught the animals to press a bar to stimulate these areas. The highest stimulus rates (between 50 and 100 percent of the animals' time) were observed when the electrodes were in the septal area, in the ventromedial and anterior hypothalamus, the preoptic area, and the cephalic tegmentum of the midbrain. In some cases, hungry rats performed better when they pressed the bar for electrical stimulation than when they pressed it to get food. We have argued that the septal area serves the estimate of odors. The influx from the lateral hypothalamus may heighten the sensitivity of this area, for to a hungry man the odor of food is doubly delectable; and the relays from the nuclei to the amygdala may induce food fantasies. In addition, relays from the ventromedial hypothalamic nuclei to the septal area and the amygdala seem to induce images of rest and relaxation. Granted some current leakage, septal stimulation seems to combine delicious fragrances, delectable images, and pleasurable relaxation—no wonder that mere food cannot compete with this attraction! The other sites of stimulation that seem to be rewarding are either connected with the septal area or with other portions of the estimative system.

Heath and his associates (1954) stimulated the septal area of psychotics and arthritics. With some electrode placements in the septal area and the rostral hypothalamus, the patients felt better, "more normal," became alert, and seemed more contented. Arthritic patients showed greater flexibility in their joints and looked and felt greatly improved. It is likely that this improvement was not merely the result of stimulating the cortical estimative system, but also, as Heath and Leach (1956) have suggested, the result of activating some enzyme system which promotes the normal functioning of this system and its connections (hippocampus and action circuit—cf. Nauta, 1956).

Heath and his associates (1954, 1957) report that psychotics show a slow wave and spike pattern in the electrogram recorded from their septal areas which is never found in normals. They found that hallucinations, often combined with rage and assaultive behavior, occur when there is increased spiking in this area with high amplitude paroxysmal spiking in amygdala and hippocampus. When an extract ("taraxein"), made from the blood of psychotics, was injected into normals, they became abstracted, slow, out of touch with the environment, and reported that they could not think, that their thoughts kept breaking off, that their mind was a blank. With higher dosage, they showed hebephrenic and catatonic symptoms. When taraxein was injected into monkeys, they also became unresponsive and catatonic and showed spikes in the electrogram of their septal areas. Extracts of septal tissue from normal animals counteracted both the behavioral symptoms and the spiking in the septal area.

Heath *et al.* (1957, 1958) suggest that there is a defect in the biochemical functioning of this area which is accompanied by spiking and psychotic symptoms. We would suppose that this defect prevents the normal use of these pathways as soon as there is such spiking, or at least makes it more difficult. Consequently, fantasies will be induced via the amygdala that have nothing to do with the needs of everyday life. The psychotic is not interested in food because images of food are not called up in the normal way by tissue needs. Instead, he is preoccupied with his fantasy life which does not let him experience hunger and does not induce normal endocrine secretion. However, there is as yet not enough evidence to allow an analysis of the effects of taraxein and its origin.

To sum up: We suggest that *hunger* is aroused by receptors in the hypothalamus that detect the lack of various substances in the blood. These receptors increase the activity of the lateral hypothalamic nucleus which sends relays to the amygdala, inducing an impulse to imagine the food needed and the action required to obtain it, and to the motor nucleus of the vagus, inducing gastric contractions which are felt as organic sensations. Consequently, an impulse is aroused to look for food and obtain it; this is mediated via the circuit from hippocampus, cerebellum, thalamus/hypothalamus to the frontal lobes. The hypothalamic route joins the fibers from the lateral hypothalamic nucleus and reinforces the impulse to action mediated by these fibers. Relays will lead to the premotor and motor area mediating the impulse to action and to the secretory hypothalamic nuclei, inducing the discharge of ACTH which leads to adrenocortical secretion promoting gluconeogenesis.

In *thirst,* the hypothalamic receptors seem to detect an increase in the sodium and chloride concentration of the blood and to relay impulses to the supraoptic and paraventricular nuclei to discharge ADH which prevents diuresis until water intake restores the normal balance. Otherwise, the neural activity resembles that in hunger. The hypothalamic tracts mediating action impulses to drink seem to adjoin those mediating the urge to eat.

Repletion, on the other hand, seems to be induced by activation of the ventromedial hypothalamic nucleus, perhaps from hypothalamic receptors detecting enterogastrone or related hormones in the blood, which are secreted when food enters the duodenum. Relays from the ventromedial hypothalamic nuclus seem to be sent to the amygdala and hippocampus, inducing an impulse to imagine arrest of action or a place to rest, and to the vagus nerve, inhibiting gastric contractions. At the same time, the distention of the stomach will be felt as slightly unpleasant. This and the changed way of thinking (from imagining food and eating to imagining rest or some other activity) will arouse an impulse to inhibit eating which is mediated via the action circuit from hippocampus to frontal lobe. The hypothalamic part of the circuit, joining the fibers from the ventromedial hypothalamic nucleus, seems to reinforce the impulse to rest and relax, which is relayed to the premotor and motor cortex. In the hypothalamus, this tract seems to run in a ventromedial plane via the septal area to the caudate and globus pallidus to inhibit extrapyramidal fibers, and via the caudate and motor cortex to inhibit pyramidal fibers.

INSTINCTIVE PATTERNS: II. THE MATING PATTERN

As hunger seems to induce the impulse to eat, so sexual tension seems to induce the impulse to mate. But sexual tension, unlike hunger, has no daily rhythm. Male mammals are ready to mate whenever they are in the presence of a receptive female, while female mammals have an estrus cycle influenced by various environmental factors, such as light, warmth, and so forth, depending on the species, and will accept the male only when in heat. In some animals, like the rabbit or pigeon, which ovulate only after sexual excitement, the sight of another animal of the same species is often enough to induce ovulation. In isolated pigeons, just the bird's own image reflected in a mirror has induced ovulation (Harris, 1948).

Apparently, seasonal factors induce the hormonal changes we call estrus, which makes the female receptive. But it is doubtful whether the mere presence of sexual hormones in the blood is enough to induce sexual activity. At least in rabbit and pigeon, the sight of others of the same species seems to be necessary for sexual arousal. We may conjecture that without it the animals cannot form the image of a mate, that in those circumstances the sexual tension induced by an increased hormone level finds no direction. Given normal experience with others of the same species, the hormonal changes at puberty will arouse or at least favor the imagining of a possible mate. The potential mate is next appraised as attractive, good for mating, which arouses, in turn, the felt sexual desire and induces the genital changes that go with it.

This would explain why there is a physiological periodicity in human females as in other mammals, yet no corresponding periodicity in sexual activity. The human being, unlike the animal, can direct imagination and does not depend on a physiological state (ovulation, menstruation) to arouse images of mate or mating. At the same time, there is a well-known increase in sexual images and sexual desire, either before or during menstruation, which corresponds to the changed hormonal state. That it is the sexual fantasy which induces sexual desire is shown also by the fact that imagining a sexual partner will induce the genital changes that go with sexual desire. Long isolation, without any opportunity to see members of the opposite sex, seems to reduce sexual fantasies and, at the same time, sexual desire, as Kinsey found in his study of long-term prisoners.

The impulse to mate. The presence of increased sex hormones in the blood (for instance, at puberty) may be detected by hypothalamic receptors in the same way as that of other blood constituents. In the case of sex hormones, the area stimulated by these receptors seems to be the infundibular nucleus in the hypothalamus (Rothballer and Dugger, 1955). Excitation of this nucleus seems to activate relays to the same structures that receive relays during hunger: the amygdala which mediates the fantasy of mate and mating by stimulation of various cortical association areas, and the hippocampus which mediates the appraisal for action and so gives rise to emotion—in this case, sexual desire. The action circuit that mediates sexual desire, according to our scheme, connects with the fibers from the infundibular hypothalamic nucleus. The combined relays to the caudate nucleus now induce the autonomic changes that go with sexual arousal, and also the impulse to mate which leads to action via pyramidal and extrapyramidal pathways. Very likely, there is also a relay to the secretory hypothalamic nuclei and to the pituitary gland, which induces the discharge of gonadotrophic hormone (GTH). We do know that in animals which ovulate only after sexual excitement a discharge of such hormones must occur to induce ovulation.

There is considerable evidence for such a circuit. Rothballer and Dugger reported that a lesion so placed that it destroyed the median eminence of the tuber nucleus in the hypothalamus and separated the infundibular stem from the hypothalamus produced retrograde degeneration of the paraventricular and supraoptic nuclei and also of the infundibular nucleus (a group of neurons in the ventromedial hypothalamus surrounding the pituitary stalk), while the ventromedial nucleus remained intact. Clinically, this patient, a fourteen-year-old boy, showed infantilism and severe diabetes insipidus with a complete lack of secretory material in the pituitary. In another case, a much larger tumor destroyed or displaced the whole hypothalamus but left the infundibular nucleus and the pituitary stalk intact. In this boy, there was no disturbance of sexual function or water metabolism. The authors concluded that the infundibular nucleus must be intact and active to initiate the endocrine changes necessary for sexual maturation.

Kucsko and Seitelberger (1955) reported a case of hypothalamic tumor which completely destroyed the hypothalamic nuclei, starting from the anterior pituitary gland and infundibulum and gradually covering the whole hypothalamus from the anterior commissure to the mesencephalic tectum. This patient, a young woman, at first complained of loss of menstruation, which was temporarily restored by estrogen injections. Later, she complained of fatigue, was difficult to arouse, and gradually became confused and somnolent until she finally died in coma. The authors pointed out that the tumor destroyed two systems, one centripetal, with impulses passing from the pituitary to the small-celled infundibular nucleus and main tuber nucleus, the other centrifugal, from the large-celled supraoptic nucleus via the supraoptico-hypophyseal tract to the pituitary gland. We would suggest that the centripetal system detects the hormone content in the blood by means of special receptors which relay impulses to the infundibular nucleus; excitation of this nucleus next stimulates the secretory (supraoptic) nucleus which induces the discharge of sex hormones from the pituitary (the centrifugal system).

The role of imagination. We are suggesting that it is the impulse to imagine mate and mating which induces the genital changes that prepare for mating and arouses directed sexual desire; we would expect that the amygdala, which seems to mediate such imagination, plays an important role. This is confirmed by a number of studies. Koikegami and his co-workers (1954), for instance, produced ovulation in the rabbit by electrical stimulation of the amygdala, and Sawyer (1955) induced ovulation in this species by injection of histamine into the third ventricle when the animal was kept under light nembutal anesthesia. Without nembutal, histamine did not induce ovulation, though it did produce persistent fast activity in the dorsal hypothalamus, the medial preoptic area, the amygdala, and hippocampus, with or without anesthesia. Apparently, histamine stimulated the amygdala and hippocampus as well as the action circuit connecting with the supraoptic nuclei, but such massive stimulation can hardly be specific enough to induce the image of mate and mating in the waking state, when innumerable sense impressions compete for attention. Under light anesthesia, when normal activity is excluded, it is much easier to induce excitation of a particular preferential pathway. Light anesthesia, as we know, does not create any interference with fantasy as expressed in dreams.

Faure (1957) found that subliminal stimulation via electrodes implanted in the corticomedial part of the amygdala and hippocampus aroused "typical olfacto-genito-sexual behavior" which was facilitated by histamine, acetylcholine, and gonadotrophic hormone. During such behavior, changes in electrical activity appeared in the records from the amygdala, hippocampus, and ventromedial hypothalamus. Stimulation of the rostral part of the infundibular nucleus has induced ovulation in the rabbit (see Kucsko and Seitelberger, 1955). Apparently, the circuit active during sexual arousal includes both the ventromedial hypothalamus and the amygdala.

Evidence seems to be accumulating that the various parts of the amygdaloid complex have different functions (because of their different connections, we think). Kaada and associates (1954), for instance, showed that there is an older anteromedial division connected with septal, preoptic, and hypothalamic regions. We would suggest that these are the areas stimulated by hypothalamic relays or by relays from the olfactory bulb. However, this older part of the amygdala is also connected with the basolateral part projecting to neocortical association areas. Because of this, impulses to imagine which are aroused by hunger, thirst, sexual tension, or olfactory sensations can produce visual, auditory, somesthetic, or motor images. Accordingly, the "attention" response, as Kaada has called a response to electrical stimulation which resembles "reactions to visual, acoustic or olfactory sensations" (1954, p. 60), can be produced from any part of the amygdala. On the other hand, autonomic responses like piloerection, salivation, micturition, or defecation have been produced only from the anteromedial division of the amygdala (Kaada and Ursin, 1957; Wood, 1958), which receives afferents from the hypothalamus and seems to be active in various instinctual activities. Bilateral lesions of the central amygdaloid nucleus (in the anteromedial division) have produced increased aggression and food intake (Wood, 1958). We would suspect that this nucleus receives afferents from the ventromedial hypothalamic nucleus and mediates the impulse to imagine resting and relaxing. If either of these structures is destroyed, there is continuous eating and increased irritability because relaxation and satiation have become impossible.

Wood also reported that bilateral lesions of the lateral amygdaloid nucleus (in the basolateral division) produced a hypersexed state eight to ten weeks following the operation. This corresponds to the behavior reported by Schreiner and Kling after removal of the amygdaloid complex (see Ch. 3), and seems to follow from the animal's inability to imagine the appropriate mate. After such lesions, he will merely obey his undirected haphazard impulse to relieve his sexual tension.

The mating pattern after decortication. Since the mating pattern, like the feeding pattern, has an instinctive basis and is found throughout the vertebrate series, and since it is always initiated bilaterally, it seems to be mediated via the extrapyramidal as well as the pyramidal pathway. This makes it possible for decorticated females to execute the mating pattern as long as the connection with the cerebellum and hypothalamus is intact. Of course, without the neocortex the activation of this pattern is possible only when a touch experience is appraised as pleasurable, i.e., good for mating. Thus the female will respond to touching of the genitals during estrus (estrus increases the blood flow and changes the sensitivity of the genital mucous membranes). This happens whether estrus occurs spontaneously or is induced by estrin injections (Bard and Rioch, 1937). But the male, who has to seek and approach a female before genital stimulation can occur, depends on memory for the appraisal that this moving thing is good for mating; he must have intact sensory and association areas. Brooks (1937) found, for instance, that mating is abolished when the olfactory bulbs are destroyed in the male after removing all neocortex. With intact neocortex, the olfactory bulbs are not necessary for mating. This means that at least one sensory avenue must be left to make it possible for the male to identify and appraise the situation and to approach the female.

Bard and Rioch (1937) found that one of their female cats spontaneously came into heat after all neocortex had been removed with part of the rhinencephalon, a little of the striatum, and the rostrolateral half of each thalamus. According to our hypothesis, this was possible because the small lesion in the striatum did not interrupt the pathway from the infundibular nuclei to the globus pallidus and secretory neurons. In the other cats estrus did not occur after removal of all cortex and much of the striatum and damage to the greater part of the thalamus. In this case, so much of the striatum seems to have been removed that the connection between the infundibular nucleus and the supraoptic nucleus was interrupted. However, these animals still showed the typical mating behavior after an injection of estrin and genital stimulation. This is what we would expect if the intralaminar and somatosensory thalamic nuclei were still intact, allowing crude sensation to be appreciated. The motor pattern would then follow reflexly via connections with the tectum, tegmentum, cerebellum, and the cerebellar projection to the red nucleus and spinal cord. Bard and Rioch found that mating behavior is permanently abolished if the brain stem is cut in a line from the caudal end of the mammillary bodies to the intercollicular level. Such a cut interrupts the connection from the cerebellum via the red nucleus to the spinal motor neurons.

Detumescence and quiescence. Sexual activity reaches a peak in the orgasm and then subsides. While such quiescence may seem no more than the end of a "consummatory response" in the mating pattern, it requires explanation just as much as does repletion after eating. The experience of pleasure in orgasm seems to be a reaction to the sensations felt during the rhythmic contraction of clitoris, vagina, and uterus in the female and of the muscles of prostate, seminal vesicles, and ejaculatory ducts in the male (cf. Fulton, 1949). Like every pleasure, this pleasure also arouses the desire to concentrate on it completely and to inhibit every other activity. Accordingly, the orgasm is followed by quiescence and relaxation. This presupposes a pathway which mediates the inhibition of muscular activity. We believe that muscular inhibition is initiated by fibers coursing in the action circuit via the ventromedial hypothalamic nucleus to the caudate nucleus, thus inhibiting pyramidal as well as extrapyramidal motor neurons. We would expect that the destruction of the ventromedial hypothalamic nucleus would result not only in increased eating (as shown in Miner, 1955) but also in increased sexual activity because relaxation and quiescence are now impossible. But there is no evidence from animal experiments to support this inference, perhaps because the ventromedial hypothalamic nucleus adjoins the infundibular nucleus and it is likely that destruction of the one would bring about damage to the other and thus prevent sexual arousal. Schreiner and Kling (1953) report that destruction of the ventromedial hypothalamic nuclei results in hyposexed vicious animals. In their experiments, two cats had developed hypersexed behavior after destruction of the amygdala, but had remained docile; after additional ablation of the ventromedial hypothalamic nuclei, the change was striking.

EMOTIONAL ACTION PATTERNS

In emotions proper (as distinguished from the desire that accompanies instinctual actions) the physiological changes follow upon perception and appraisal instead of preceding them.

Anger. Reactions of anger with defensive posture, lashing of tail, extension of claws, hissing, spitting, retraction of ears have been induced in cats by electrical stimulation of the periaqueductal gray matter of the midbrain, the perifornical area of the hypothalamus, and the medial preoptic and ventral septal region. These reactions ended in well-directed attack upon any object near (Hess, 1956; Hess and Akert, 1955). Rage has also been produced by stimulation of the amygdaloid complex, of various limbic areas, and of the hippocampus (Kaada *et al.,* 1953), as well as by stimulation of the fastigial cerebellar nuclei (Zanchetti and Zoccolini, 1954).

Apparently, anger or rage is mediated via the action circuit, which relays impulses from limbic areas to hippocampus, cerebellum, thalamus and hypothalamus, caudate nucleus, and frontal lobe. The tract from the hypothalamus to the caudate nucleus seems to run more dorsally than the fibers mediating relaxation, since damage to the basal aspect of the brain rostral to the optic chiasma, and of the posterior orbital surface, has evoked attacks of rage (Spiegel *et al.,* 1940), which implies that the tract mediating rage must be intact, the pathway mediating relaxation, interrupted.

The urge to fight seems to be experienced when relays from the caudate nucleus and ventral thalamus arrive in the premotor cortex, thus mediating the impulse to attack with teeth and claws. The autonomic changes accompanying anger (change in blood pressure, heart rate, etc.; see Ch. 7) seem to occur when impulses arriving in these cortical regions stimulate sympathetic motor neurons via pyramidal and extrapyramidal connections. In addition, these connections stimulate the secretion of noradrenaline from the adrenal medulla. We shall see in Chapter 7 that the secretion of noradrenaline is increased during anger.

The secretion of saliva and gastric hydrochloric acid seems to be stimulated at the same time. French and associates (1954) have found that daily electrical stimulation of medial points in the hypothalamus in an area from preoptic to postmammillary region produced gastric ulcers in monkeys within thirty days. These monkeys became irritable and agitated and showed acute malaise. This would suggest that anger and worry may stimulate the action circuit in a similar way to wanting food and eating it, at least in man and monkey.

As soon as a man appraises something as annoying and feels anger (whether he expresses it or not), he immediately imagines what to do to fight effectively, even if all he does is use bad language. Such motor fantasies seem to be mediated via the amygdala and the frontal association areas, and reinforce the anger and attack as well as directing it. Finally, anger and attack are registered as motor engrams and so facilitate future anger and future aggressive action. This promotes not only the gradual development of an emotional attitude (irritability), but the gradual tracing of the marks of anger in face and posture.

Fear. The impulse to escape is similarly organized into a coordinated action pattern. Hess found that running and escape was induced by electrical stimulation of a region about two millimeters from the midline in the posterior hypothalamus and subthalamus, extending backward and upward to the pretectum and tectum, a region close to the pathway for anger (see Gloor, 1954). In fact, this "flight" region seems to extend not only backward but forward to the caudate nucleus and the frontal lobe, for Ingram *et al.* (1954) report that stimulation of the caudate and dorsomedial thalamic nuclei have produced searching and apprehension in animals. It seems reasonable to postulate a "fear" circuit that can be excited by an appraisal that "this is dangerous, to be escaped," and runs from the hippocampus via the lateral ventral thalamic nucleus to the premotor and motor areas, mediating the urge to flee; and via the anterior and medial ventral nuclei to the prefrontal cortex, serving the registration of the flight pattern. The autonomic changes can be touched off via the hypothalamic branch of the action circuit (which includes the adrenaline-sensitive fibers—see Ch. 5, Drug Effects), running over the caudate nucleus to the promotor and motor areas, but connecting also with extrapryamidal pathways. Since electrical stimulation of "flight" points in the hypothalamus results in increased adrenaline secretion (Folkow and Von Euler, 1954), relays from the hypothalamus seem also to connect with sympathetic fibers to the adrenal medulla. Finally, fear is accompanied by increased adrenocortical secretion (see Ch. 8), induced by the discharge of ACTH from the anterior pituitary gland—a discharge apparently initiated by relays from the caudate nucleus via the pallidohypothalamic tract to the hypothalamus and pituitary gland.

Our conception of the circuit activated in fear can explain some puzzling findings. W. W. Roberts (1958) reported that electrical stimulation of the posterior hypothalamus of cats, just rostral to the mammillary bodies, produced a "flight" reaction with alerting, looking around, searching, and attempted escape. The cats quickly learned to *escape* such stimulation by climbing through an opening to the other side of the apparatus, but they did not learn to climb through the opening to *avoid* stimulation. Cats given an electric shock through the bottom of the cage or stimulated in other parts of the brain (mainly in the somatosensory areas) learned not only to escape such stimulation but to avoid it before it was given.

It would seem to us that a stimulation of "flight" points in the posterior hypothalamus induced a generalized impulse to run. The cats followed this impulse as long as they felt it. When the current was turned off, they no longer felt the impulse and stopped. They could not learn to avoid the stimulation because they never felt an impulse to escape until the current was turned on. When other points in the brain were stimulated (particularly in the somatosensory areas), there was an appraisal for action and emotional arousal. Stimulation appraised as bad, to be escaped, was *feared* and could be avoided.

Stimulation at points along the last link of the action circuit, between brain stem and frontal lobe, will produce a felt impulse to action. This impulse will be appraised (hence, the looking around) but given in to when the stimulation continues and the impulse continues to be felt. Since the animals see nothing that could prevent running, they follow their impulse and run.

THE REACTION TO FEELINGS

Feelings as well as emotions may arouse a desire for action. An intense light is avoided by shutting the eyes; an unpleasant sound may drive a person out of a comfortable room; and too warm or too cold weather arouses a variety of desires, for anything from a long cool drink to a mink coat. Most feelings present no special problem because the sequence from perception to appraisal, feeling, and the desire for action, is much the same as in emotion. But the feelings of discomfort that come with intense warmth or cold are in a category apart. We shall try to pinpoint the problem and attempt a reasonable solution.

Discomfort from heat or cold. The discomfort from visual, auditory, or touch sensations is felt immediately. It is the result of an appraisal of sense impressions of which we are aware. But sensations of warmth or cold seem to affect the body as a whole before we can appraise them as pleasant or unpleasant. Now it is generally accepted that there are warmth and cold spots in the skin just as there are touch spots. But the sensations they mediate involve autonomic changes long before they become unpleasant. In fact, both warmth and cold are usually noticed first because of the changes they produce: the skin flushes or pales, becomes warm or cold, we sweat or shiver. This would indicate that temperature changes somehow affect the organism as a whole, and it is this effect that is felt as either pleasant or unpleasant.

We believe that the fibers that mediate warmth and cold are connected with vasomotor fibers and induce reflex dilatation (during warmth) and reflex constriction (during cold). The connecting fibers apparently run in the sympathetic chain for part of their course and mediate both local and intersegmental vasomotor reflexes (Cooper and Kerslake, 1955). These reflexes seem to reinforce the sensations of warmth and cold and represent the effect of such stimulation on the body.*

This effect of warmth or cold is next appraised as either pleasant or unpleasant. The vasoconstriction induced by cold (e.g., when the hand is put into ice water) is felt as unpleasant, while the later vasodilatation is felt as pleasant. But the extreme vasodilatation that comes from lengthy exposure to the sun is soon felt as unpleasant; and the vasoconstriction produced by a cold shower will then be very pleasant. Whether a given effect of warmth or cold is appraised as pleasant or unpleasant depends on the state of the organism as much as on the stimulus. In the heat of summer, a cold drink or shower is pleasant. In winter, in an unheated room, it would be decidedly unpleasant. Such changes in feeling can only be explained as the appraisal of an effect that is superimposed upon an organismic state (the effect of earlier stimulation) which it either counteracts or intensifies.

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* We venture to suggest that the fibers mediating warmth and cold that run in the lateral spinothalamic tract close to the pain fibers are actually fibers of the estimative system. This would account for the fact that warmth or cold may be felt as comfortable or uncomfortable, pleasurable or painful, rather than as warm or cold, when the sensory thalamic nuclei or the somesthetic cortical areas are destroyed (see Head and Holmes, 1911). This would mean that some beta fibers from the warmth and cold receptors should run together with the cutaneous fibers in the dorsal funiculus to the sensory thalamic relay nuclei and the somesthetic cortex, to inform us of the *quality* of warmth or cold. To date, such connections have not been reported.

Reaction to heat. During hot weather, we are flushed, feel uncomfortably warm, and perspire a great deal. We also feel a desire to rest and cool off, whether by taking a swim or a cool drink. Animals also seem to be uncomfortable and look for a shady place. Dogs and cats sit up and begin to pant.

These symptoms can be reproduced by electrical stimulation of a region from the septum and preoptic area through the lateral and dorsal hypothalamus to the central gray in the rostral midbrain. Such stimulation has produced salivation and panting in cats, with the typical sitting up (Hess, 1949). Andersson *et al.* (1956) have found that stimulation of the dorsal half of an area between the anterior commissure and the optic chiasma, extending laterally to the medial edge of the internal capsule, produces panting and vasodilatation in goats. This pathway seems to be active during heat.

As soon as we have succeeded in cooling off, or as soon as we have become accustomed to the heat, the flush and perspiration subside. In animals, panting stops. This organismic reaction to the effect of heat seems to require a central pathway also, if we may judge from recent reports. McCrum (1953) found that an area close to the midline in the hypothalamus, from the anteroventral part of the tuber cinereum to the anterior commissure, but extending more laterally in the anterior hypothalamus, seems to be necessary for maintaining normal body temperature in a hot room or after morphine injection. This pathway seems to lie in a ventral and medial direction from the pathway mediating heat. When toxic substances course in the blood, both these pathways may be activated; thus we get the flush and high temperature of fever, alternating with chill and shivering.

This organismic reaction to the effect of heat has been observed by Kundt *et al.* (1957). Warming of the paws of rabbits produced first vasodilatation in the ears (effect of heat) and then, after a few seconds, a drop in hypothalamic temperature of 0.05° C.; when the hypothalamus was cooled locally in another animal by 1° C. or less, marked vasoconstriction appeared in the ears, as well as shivering and a rise in rectal temperature. This seems to indicate that the effect of heat is counteracted by a drop in hypothalamic temperature (probably produced by constriction of hypothalamic blood vessels), which is soon followed by general vasoconstriction. We believe that the pathway serving this organismic reaction relays impulses to pyramidal and extrapyramidal pathways connecting with the sympathetic system.

Reaction to cold. When we feel cold, we become pale, shiver, and feel most uncomfortable. We feel a desire to do something to warm up, to take a brisk walk, put on an extra sweater, or turn up the theromostat. Animals also show signs of discomfort, shivering, and piloerection. These effects of cold can also be produced by electrical stimulation of the lateral hypothalamus, which induces vasoconstriction (Fulton, 1951).

As soon as we begin to get warm, shivering subsides and pallor gives way to natural color. This seems to imply a secondary vasodilatation that is increased on acclimatization to cold (see Ch. 8). It may come when we are moving about but can occur even when motion is prevented. Sherwood and associates (1954) have found that normal temperature can only be maintained in the cold if an area is intact that extends from the superior border of the optic chiasma through the dorsal part of the lateral hypothalamus toward the midbrain. The organismic reaction to cold, which restores normal temperature, is apparently mediated via a dorsolateral hypothalamic pathway, which relays vasodilator impulses to pyramidal and extrapyramidal tracts connecting with the sympathetic nervous system. This vasodilatation seems to warm first the hypothalamus and finally the rest of the body (see Ch. 7). Kundt and coworkers (1957) have found that cooling the paws of rabbits first produced vasoconstriction in the ears and, after a few seconds, a rapid rise in the hypothalamic temperature (by 0.06° C.); and Von Euler (1950) has reported that local heating of the anterior hypothalamus produces panting in the animal, and also slow electrical potentials from this region. We have noted that slow potentials induce muscular relaxation, which agrees with the fact that muscles relax during warmth.

We are suggesting that the organismic reaction to both heat and cold is initiated from the thalamic estimative system via relays over the midbrain and the hypothalamus. When the midbrain was completely transected in dogs, the temperature of these animals was about 5° C. higher than room temperature, and they were unable to counteract either heat or cold. When the room temperature rose above 35° C., their temperature rose accordingly and they died in hyperthermia. When it fell below 30° C., animals with transected midbrain died of depressed circulation (Andersson *et al.,* 1957). Only the vasomotor reflexes that mediate the environmental effect on the body were active in these animals while the organismic reaction was prevented. The same phenomenon can be observed in the limbs of paraplegic patients below the cord transection (Wyndham, 1955).

Intense local cooling seems to produce a local vasoconstriction which cannot be counteracted by the organismic reaction. As a result, it prevents the conditioned vasoconstriction to a sound or light announcing an electric shock (Shmavonian, 1959). This would indicate that the effect of *pain* also includes vasoconstriction, and further, that this constriction, like the muscular contractions that result in withdrawal, can be induced by the expectation of pain to come, i.e., as the sequel of a conditioned stimulus.

Part of the organismic reaction during cold is the secretion of ACTH and TSH (thyroid-stimulating hormone) from the anterior pituitary gland, which stimulate the adrenal cortex to secrete adrenocortical hormone and the thyroid gland to secrete thyroxine. But hamsters, either in the hibernating or the nonhibernating state, do not secrete ACTH and TSH during cold (Deane and Lyman, 1954). For these animals, cold is the signal to hibernate and not something that is uncomfortable or damaging: hence no ACTH secretion. And for hibernation they do not need to restore normal temperature: hence no TSH secretion. However, even without TSH secretion, these hamsters do regain their normal temperature if the environmental temperature rises again. This would indicate that thyroid secretion is essential only for retaining normal temperature during cold. The fact that only a felt stress induces ACTH secretion would support our suggestion that the organismic reaction is initiated by the estimative system. This is also implied in Beattie and Kerr's report (1936) that the caudal half of the medial thalamus must be intact for shivering to be induced by cold. We know from experience that shivering begins after we have begun to feel uncomfortable.

We propose to examine the peripheral effects of emotions in Chapters 7 and 8.

DELIBERATE ACTION

Now that we have discussed instinctive and emotional action patterns as well as the overt reactions to feelings of pleasantness and unpleasantness, let us look at deliberate actions, actions that seem to be prompted neither by emotions nor by feelings. A child may practice the piano without any desire to do so. Even if he likes to play, there is no immediate pleasure attached to hitting one key after the other. The pleasure comes with the virtuosity of his performance, or the conviction of something well done, or the joy in sound and harmony. In any case, the child is not attracted to each finger movement before he makes it, nor does he follow an instinctive pattern automatically. The decision to practice, to play well and with expression, is a rational decision. The emotion he expresses during his performance is an emotion that flows from an aesthetic appreciation of the music, not from an intuitive judgment that the finger movements are pleasurable.

In deliberate actions (and they comprise the large majority of our daily activities) we must depend on a judgment that is not intuitive to arouse an impulse to do something that may or may not be pleasant. Whatever may be the explanation for such rational judgments and deliberate actions, it is such judgments and actions that distinguish man from the brute. Traditionally, rational judgment and deliberate action have been explained as *activities of the mind, depending on consciousness.* Whatever the explanation, it will lead us sooner or later to the deeper problem of the relationship between mind and brain, between consciousness and the neural paths that mediate it.

Consciousness, mind, and brain. Objective psychologists may scorn this problem (see Hebb or Lashley, in: Delafresnaye, 1954) and philosophers of science may seek to convince us that it is a semantic pitfall (see Ayer or Ryle, in: Laslett, 1950); but neurologists and neurophysiologists are seriously troubled by it. Thus Penfield says:

> What is the real relationship of this mechanism [the brain] to the mind? Can we visualize a spiritual element of different essence capable of controlling this mechanism? When a patient is asked about the movement which he carries out as the result of cortical stimulation . . . he knows he did not will the action. He would agree that something else finds its dwelling-place between the sensory complex and the motor mechanism, that there is a switchboard operator as well as a switchboard. (In: Laslett, p. 64)

And Eccles speculates that the mind may induce microscopic spatiotemporal patterns of activity in the brain and says: "It would appear that the brain is the sort of machine a 'ghost' could operate, if by 'ghost' we mean an agent whose action has escaped detection even by the most delicate physical instruments" (1953, p. 285).

The cardinal difficulty in both these statements is the assumption that the brain is completely separate from the mind, that the nervous activity in the brain is entirely divorced from the experience that depends on it. Both authors assume that sensory impulses end up at some "highest level" of the brain (for Penfield, the brain stem "centrencephalic system"; for Eccles, the cortex) and that there they are received by the mind which in turn sets the motor system in action.

Perhaps this assumption is the result of decades of anatomical and neurophysiological experimentation with the dead or anesthetized brain. Such experiments have made it easy to believe that the brain consists of a feltwork of intricate wiring which merely needs to be activated to produce a given psychological experience. Similarly, psychologists have continued to hold the stubborn belief that psychological activity can be predicted once the neurological pathways are accurately mapped.

Eventually, neurologists found that electrical stimulation of the human brain does not produce the same experience as do neural impulses arriving in the same area. Conscious patients report that electrical stimulation of the motor cortex (during brain operations) does not produce the experience of having willed the motion; and stimulation of the sensory cortex does not produce the same experience as does sensory perception. Accordingly, Penfield recognizes that psychological activity is never exactly duplicated by stimulating brain cells or tracts, and suggests at least half seriously that there must be some "spiritual element" operating these relays. Compelled by the same evidence, Eccles is trying to show that the brain is the kind of machine such a ghost could operate.

Brain and person. But the living brain is an integral part of a living being, it is not simply an intricate electrical system. The brain does not produce sensations, images, or thoughts, *we* do. When we sense, imagine, think, certain pathways are active and communicate activity to a widespread area; but *we* sense, *we* imagine and think, not our brains. Similarly, it is not our muscles that produce a movement, *we* do. We intend to move in a certain direction, and while we are so intending (or together with our intention), muscles become organized into a pattern and in a sequence that comes out as movement. We do not intend to move *muscles*. We may not even know which muscles should be contracted and which relaxed for a given movement. But intending to move in a particular direction, the appropriate muscles are activated in the manner we have attempted to describe in Chapters 3 and 4.

Only in such a way can we conceive of a "psychosomatic unity" without contradiction. So long as we assume there is one thing: the body with its brain that behaves just like any mechanical system; and another thing: the self (or mind) that has experiences produced in some way by the physical system and that can move this system in turn, we will always have two things rather than a unit.

It does not help much to say that there is only one thing, the body with its brain, and that what we call "mind" is merely the sum of all psychological processes, presumably also produced by the body (see Cobb, 1952). This expedient leaves us with two types of processes that are experienced quite differently, yet are apparently produced by the same source. I *have* a toothache or an upset stomach, good or poor digestion or circulation. But *I* think, remember, imagine, act. *I have* a body; but *I myself do* the thinking, deciding, acting, experiencing. Apparently, some of my experiences merely happen to me; but others, I know, take their origin from me. It is difficult to conceive that something I *experience* (my body) should produce the *I* that *has* the experience.

The person as agent. The solution we propose is simple, though by no means new. We conceive of the living being as an actor or agent that acts in, through, or by means of the organized body. This body is a distinguishable, but not distinct, part of the living being. *I as a living being* initiate both biological and psychological activities. As long as I am alive, I live in my muscles, brain, and tissues just as much as in my perceiving, thinking, or remembering, and in my decisions for action. But physiological activities go on without my personal attention, so to speak, while psychological activities, and particularly goal-directed action, require that I be aware of the things around me and that I be able to deal with them in some way.

Consciousness and the estimative system. The activity of a living sentient being is impossible without a nervous system or its equivalent. But this activity is not produced by the nervous system, any more than a telephone conversation is produced by the cables that carry it.

At every level of being, an individual can be said to be conscious only when he is aware of what goes on around him and how it affects him. The *quality* of sense impressions is important for gauging their *effect* on him. Without the possibility of estimating the effect of the environment, psychological activity would be impossible. We have argued that the effect of stimulation can be gauged via the *estimative system* which receives fibers from every sense receptor. When this system is interrupted so that impulses can no longer be relayed to the thalamus and limbic cortex (e.g., when the medial thalamus is damaged or the brain stem reticular formation is cut), appraisal of the environment is no longer possible. There may be an awareness of sensations; but without being able to appraise them in any way, man or animal cannot act and cannot even pay attention to them. The normal functioning of the estimative system seems to be necessary for a mere awareness of something, while full consciousness demands the normal functioning of the memory, imagination, and action circuits as well.

Physiological activities can go on without awareness as long as man or animal is kept alive, for these functions do not depend on conscious appraisal but on brain stem reflexes. Even these seem to require a registration of the effect of stimulation which seems to be mediated by the fine fibers of the estimative system. Bodily tissues, including brain, nerves, and muscles, and the functioning of various organ systems can be maintained without the conscious appraisal that allows the identification of stimuli for appropriate action. But the effect of high or low blood pressure, of inspiration and expiration must be gauged in some way before the proper reaction can be initiated. This is done, we believe, via the subcortical and subthalamic portions of the estimative system.

The "unconscious." What is called the "unconscious" is not identical with this primitive appraisal, for it presupposes awareness. The unconscious impulses described by Freud and other psychoanalysts influence action only by swaying the estimate of the situation that leads to action. To be influenced in this way, a person must be aware of something, even if only of the imaginary scenes that are the fruit of his emotional preoccupation. What is usually meant by the unconscious seems to be the combination of unacknowledged desires and unrecognized emotional attitudes that influence a man's rational judgment and urge him to act contrary to it.

Conscious action in man and animal. There is a difference between animal and human actions. An animal is guided to action by an emotional impulse, based on the estimate of the effect of this thing here and now. The human being judges not only on this basis but also on the basis of abstract and long-range considerations. An animal or a young child can appraise a particular kind of food as good or bad, and select an adequate diet accordingly. Animals and young children may also be tempted by specially tasty morsels to overeat, or to acquire food habits that result in some nutritional deficiency. In both cases, they act according to sense judgment, the sheer intuitive appraisal of what is good or bad here and now. But the older child and the adult can decide to abstain from certain foods (and do so) because they want to avoid undesirable consequences (e.g., overweight) or because their health requires dieting. In such a case, the human being employs reflective judgment in addition to sense judgment. He must initiate an action impulse that is contrary to his immediate preference by considering the rational alternative, finding it good and thus wanting it. In practice, this requires turning his attention to other aspects of the situation, imagining possible consequences so as to decide on the right action, and, finally, choosing the action and so arousing the action impulse that will bring it about.

Voluntary attention. Here we come to grips with the psychological aspect of self-determination or freedom of choice. Psychologically speaking, it means that we can do what we judge to be appropriate, even though it be neither pleasurable nor appealing. To make a rational judgment undeterred by an emotional attraction that is pulling in the opposite direction, a man must be able to direct his attention to an alternative course of action. Now we have suggested that attention is a desire to know something, and is appetitive rather than cognitive in nature (cf. Vol. I, Ch. 10). It is the result of an intuitive or reflective appraisal that this is good to know, and it initiates an impulse to look, remember, imagine, or identify this thing. Intuitive appraisal is followed by spontaneous (involuntary) attention, while reflective appraisal leads to voluntary attention, a deliberate looking and investigating.

Obviously, if an emotional attraction is either very strong or comes upon us suddenly, it may lead to action before we have had the time or inclination to direct our attention to its less desirable aspects, that is, before we have reflected upon it. The psychopath, apparently, has no desire to know whether anything he wants to do is either suitable or desirable. He acts without bothering to appraise his action.* But even the psychopath may do what he does for rational (though not reasonable) motives rather than through sheer emotion as, for instance, the boy who forges a check to pay for flying lessons. The normal adult, by and large, pays due attention to the consequences of an intended action and, as a result, may delay or inhibit it. He may even decide on a course of action that entails unpleasantness and suffering, not because he is a "moral masochist" and derives libidinal pleasure from it, but because he is willing to put up with suffering for a good reason.

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* A man may deliberately disregard the legal, social, or personal consequences of his action, or plan to evade them. In that case, he is a criminal rather than a psychopath. A psychopath may find it difficult to appraise his intention, either because he is unable to plan effectively or because he cannot delay his action until he has reflected upon it. In such a case, there may be a defect in motor imagination or intuitive appraisal of action impulses; there is a possibility that the functioning of the prefrontal, premotor, or anterior cingulate areas may be defective. This would explain why the psychopath always recognizes that he has done something wrong, why he sincerely promises to do better, yet always repeats his mistakes.

Imagination and concepts. Turning our attention to what we want to know for action means calling up images of possible actions and their consequences. These images need not be particularly vivid or compelling. The experiments of the Würzburg school have made it clear that much abbreviated images are used in thinking, but they have also shown that thinking cannot be explained by images alone. The concepts we use in thinking are not exhausted by the images we use to recall them. Every concept has a name, but the name is not the reality to which we refer when we use such a name. The word "liberty," for instance, refers to something abstract that may be *symbolized* by the Statue of Liberty, the American flag, or even by a nightcap; but it is not any of these things, nor is it the printed letters that make up the word, nor the sound that is used to pronounce it. What it refers to may be delimited by a definition, but the concept is understood even without definition by anyone who knows the English language. If a concept were no more than the word used to refer to it, as is sometimes affirmed, there would be no difficulty in explaining it. Similarly, if thoughts were no more than "subvocal talking," there could never be any difficulty in communicating our thoughts.

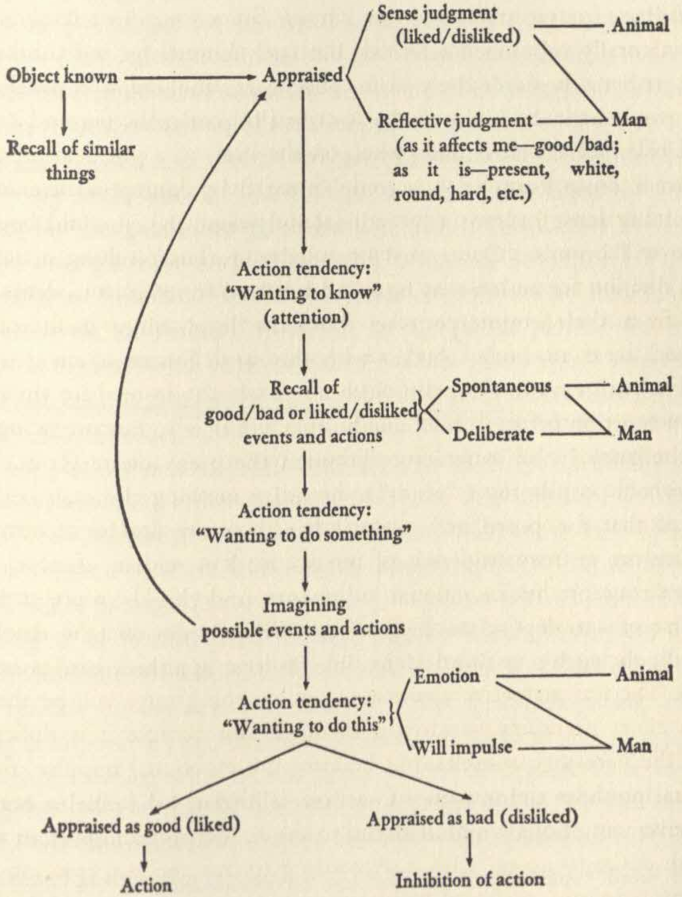
We use words to convey concepts and thoughts; and words can be recalled as visual, auditory, or motor images. We have shown in Chapter 2 that sensory images seem to be localized in separate association areas for the various sense modalities. But there is no evidence that the concepts recalled by means of such sensory images are similarly localized. For instance, after damage of the visual association areas a patient may be unble to read, yet still understand what is said to him. This means that though the visual images needed for concepts (the written or printed words) cannot be used to interpret what he sees, he must still have these concepts at his disposal or he could not understand what he hears. In fact, both the visual and auditory association areas may be damaged, yet the patient may still recognize words by tracing them (cf. Nielsen, 1946). As long as subcortical connections are intact, he apparently is still able to deal with abstract concepts on the basis of such images as remain at his disposal. This seems to show that a concept is not bound to any one locus in the brain, that it can be formed and used as long as sensory images remain in at least one of the sense modalities that are commonly used for conveying concepts, and as long as the appropriate association area remains connected with the amygdala, hippocampus, and action circuit.

Deliberate action and the action circuit. Since images of remembered, planned, or expected events provide the raw material for our thinking, it seems reasonable to suppose that conceptual thinking and intellectual judgment use the circuits we have described for remembering and imagining. Deliberate action is undertaken on the basis of a practical judgment of what is the appropriate thing to do. Since this judgment is the extension of an immediate intuitive appraisal (sense judgment), it could be mediated over the same circuits as sense judgment. The resulting action impulse, like the action impulse resulting from sense judgment, seems to be similarly mediated by neural relays from the hippocampus to the cerebellum and the frontal lobes. Such an impulse to deliberate action is usually called a "will impulse" to distinguish it from the immediate unwitting attraction to something emotionally appealing. The human being is a unit; he cannot will something without having any desire for it. What is reasonable is also desirable, even though something else may be vastly more attractive and not very reasonable. There are degrees of attraction and degrees of reasonableness of an action; but seldom, if ever, is an action dictated either by emotion or by reason alone. The more attractive a course of action, the more effortless will it be because the emotional impetus, springing uncalled from the intuitive appraisal, carries us into action. The less attractive a course of action, the greater will be the conscious effort necessary to carry it through, not because it is difficult to move the necessary muscles, but because the emotional impulse, the desire that provides the tendency to action, is almost too feeble to organize the movement. Thus we must attend to the action over and over to arouse enough desire to go on with it. According to our analysis, emotion without any reflection will lead to impulsive acting out, to an action that is hardly human, while a will impulse without any desire could not produce action at all.

The diagram below shows how perception leads to action. When we first notice and appraise something, we feel an impulse to know more about it. We attend to it, recall similar things, their effects and our reactions.

Appraising them, we now want to do something to cope with this thing. Imagining possible actions and their effects, we next want to do something definite—which (in man) is both an emotion and a will impulse. When this action tendency is found good, we act.

To sum up: Deliberate action, whether eating or playing the piano, does not need a "ghost" pulling strings somewhere in the brain. Rather, our


Sense judgment (liked/disliked)
Animal
Object known
Appraised
Reflective judgment
Man
(as it affects me—good/bad; as it is—present, white, round, hard, etc.)
Recall of similar things
Action tendency: "Wanting to know" (attention)
Recall of good/bad or liked/disliked events and actions
Spontaneous
Animal
Deliberate
Man
Action tendency: "Wanting to do something"
Imagining possible events and actions
Emotion
Animal
Action tendency: "Wanting to do this"
Will impulse
Man
Appraised as good (liked)
Appraised as bad (disliked)
Action
Inhibition of action

PSYCHOLOGICAL ACTIVITIES FROM PERCEPTION TO EMOTION AND ACTION

Since the human being can always direct his attention to different aspects of the situation and different possibilities of action, he can prolong the process of deliberation indefinitely. To act, he must concentrate on one course of action found suitable and *exclude every other:* then he will want to do what he has found suitable and his wanting will carry him into action. Phenomenologically speaking, the act of choice consists in concentrating on one particular action impulse.

perception, appraisal, and decision for action (which is a psychological intention as well as a relay of nerve impulses in the brain) organize the action pattern and initiate the action impulses that eventually innervate muscles and bring about movement. There are other activities that are organized for us, which need no such intention, e.g., vegetative functions and physiological reflexes. Even these are influenced continuously by the ongoing psychological activity (which is also brain activity). From a knowledge of nerve tracts and tissue functions we may be able to predict these naturally organized activities, but such knowledge will not enable us to predict a person's decision for action, for the simple reason that it is his psychological activity that organizes the particular pattern of relays in the brain which is required by a given action.

In man, sense knowledge is complemented by conceptual knowledge, appraisal or sense judgment by rational judgment, the emotional impulse by the will impulse. Conceptual knowledge, rational judgment, and rational decision for action may be called spiritual functions * to distinguish them from their counterparts on the sense level. Since these spiritual functions seem to work together with the sense functions, they can use the same pathways in the brain. Such a hypothesis accounts for the difference in function between man and animal, yet does justice to the remarkable similarity in brain structure between the great apes and man, and this without postulating a "ghost" to organize nerves and muscles in man. Granted that the postulated spiritual functions are not localized in the brain, as are sensory impressions, images, and memories, yet the ability to form concepts, make rational judgments, and choose an action by reflection seems to depend on the presence of the circuits we have described. Accordingly, such a notion is defensible, at least as a theoretical construct.

CONCLUSION

In tracing these action patterns, we have elucidated the relation between instinctive, emotional, and deliberate action, which is important for a

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* The word "spiritual" should be less anxiety-arousing to the psychologist than the word "ghost," used by Eccles. What is implied by the term "spiritual" does not necessarily connote anything more supernatural or superstitious than the psychological difference actually existing between a man and his dog. It seems odd that in English the word "spirit" or "spiritual" should have fallen under a scientific taboo, while in the other Teutonic languages as well as in the Romance tongues, the corresponding words have a respectable scientific usage.

theory of *motivation*. We have suggested that *instinctive* action requires the experience of some physiological need which triggers off the image of an object that is appraised as good and can fill the need; this leads to an impulse to find and possess the object. *Emotional* action requires merely that something be known and appraised as good for a particular action. The emotion is instinctive in the sense that man or animal is inherently capable of feeling it, but the action is emotional and not instinctive because there is no physiological need that has triggered off the emotion. *Deliberate* action, finally, is the result of reflective as well as immediate appraisal; and reflective appraisal depends on man's ability to direct his imagination and thus to explore various alternatives of action.

Our analysis of the sequence from perception to emotion and action and our tracing of the circuits mediating it seem to throw light not only on motivation, but upon other persistent psychological problems. *Perception,* for instance, can now be analyzed and described. It requires sense experience that must be identified by remembering similar situations and their effect on us. Personality factors in perception play a role in this process of remembering and comparing, which gives ample opportunity for the selection and interpretation of immediate experience according to individual preferences.

Learning also stands to profit from such an analysis. Essentially, learning means acquiring either new knowledge or new actions (see Arnold, 1954). New *knowledge* implies understanding something new by fitting it in with what we already know and thus recalling it easily. It requires first of all a desire to know (a "set" to learn). Next, the learner must compare the thing to be learned with what he already knows, reflect on it, and imagine how the new and the old are related. This will enable him to refocus quickly on the new information. When such comparing and reflecting is impossible (e.g., in learning nonsense syllables), he must acquire the skill of recalling what he has heard or read by repeatedly trying and having his effort corrected. Acquiring new knowledge implies activity, namely, the effort of refocusing on the registered sense impressions. We have seen that this effort is mediated by neural impulses from the hippocampus via the midbrain and thalamus to the association areas (see Ch. 3).

Acquiring a new *action* means setting up a new goal that is wanted, estimating the action that will lead to it, remembering the result of similar actions, and imagining how they should be changed to lead to the desired end. The animal does not set up a new goal for himself but finds it ready-made, naturally determined (see Arnold, 1954). The experimenter merely forces the animal to learn new actions to obtain what he wants (food, mate) or to avoid what he dislikes (electric shock). In man or animal, learning a new action is mediated over the same circuits as we have mapped out for remembering and identifying the object, wanting it, appraising the necessary action, imagining alternatives, and finally choosing one and acting upon it.

Conditioning is merely a variation of learning. *Classical* conditioning requires that the individual recognize the conditioned stimulus as belonging to or announcing the unconditioned stimulus: the buzzer as announcing the meat. It requires remembering that meat has followed the buzzer in the past and that it was good to eat; it also requires expecting the meat to come as soon as the buzzer is sounded. The autonomic change (salivation) is initiated together with the emotional action impulse (wanting the meat); it is mediated via the action circuit from the hippocampus to the hypothalamus, premotor and motor cortex, and the motor nucleus of the vagus. *Instrumental* conditioning requires the appraisal that something must be done to get the wanted food or avoid the feared shock. Like motor learning, it requires appraisal, memory, imagination, and is mediated via the action circuit.

While the cognitive theory neglects the action necessary for learning, and the S-R learning theory neglects the necessary knowing and appraising without which there could be no action, our hypothesis includes both knowing and acting, and specifies wanting as the necessary link between them. As Scheerer predicted, our phenomenological analysis has shown that emotions are "the unique expressions of the unity of cognitive and conative functioning" (1954, p. 122). Without knowing something, there can be no wanting; but wanting implies an imagined goal toward which it urges, and thus both motivates and initiates the action. Emotion and conation cannot be separated, though the human being, as we have seen, can supplement the emotional action tendency by deliberate effort. Our phenomenological analysis has led, in addition, to an identification of the brain structures that are active in knowing, wanting, and learning.

7. PHYSIOLOGICAL EFFECTS OF EMOTION

We have seen that emotion is essentially an urge to action, induced by the appraisal that something is good or bad for us and demanding a particular way of dealing with it. Some emotions (love, anger) tend toward their object, others (dislike, fear) tend away from it. Still others (joy, delight) do not urge to action but induce relaxation, making us content to rest with what we have and prize. We have argued that the appraisal which arouses an emotional action tendency initiates a whole pattern of neural impulses that include impulses activating voluntary and involuntary muscles (emotional action and emotional expression) and impulses inducing autonomic and endocrine changes. Now we will discuss in detail just what the physiological changes are that are produced in emotion.

CANNON'S EMERGENCY THEORY OF EMOTION

The connection of emotion with various autonomic changes was first systematically investigated by Walter B. Cannon. Like his predecessors in the field of emotion, Darwin and James, Cannon assumed that the physiological changes occurring during emotion will facilitate emotional action. Cannon's investigations were restricted to animals: cats and dogs tied to the animal board or enraged by pinching their tail; or cats confronted by a barking dog. The most impressive aspect of emotional behavior seemed to be the great expenditure of energy during fight or flight. Accordingly, Cannon suggested that emotion serves emergency action. He ascribed the physiological changes he observed to sympathetic excitation and adrenaline secretion, and concluded that the function of the sympathetic nervous system is to prepare the body for emergency action:

> The emotional responses . . . may reasonably be regarded as preparatory for struggle. They are adjustments which, so far as possible, put the organism in readiness for meeting the demands which will be made upon it. The secreted adrenin cooperates with sympathetic nerve impulses in calling forth stored glycogen from the liver, thus flooding the blood with sugar for the use of laboring muscles; it helps in distributing the blood in abundance to the heart, the brain, and the limbs (i.e. to the parts essential for intense physical effort) while taking it away from the inhibited organs in the abdomen; it quickly abolishes the effects of muscular fatigue so that the organism which can muster adrenin in the blood can restore to its tired muscles the same readiness to act which they had when fresh; and it renders the blood more rapidly coagulable. . . . In short, all these changes are directly serviceable in rendering the organism more effective in the violent display of energy which fear or rage may involve. (1939, p. 228)

Thus Cannon claims that emotion (as exemplified by fear and rage) energizes the organism. Sympathetic excitation and the secretion of adrenaline in emotion produce physiological effects which account for the increase in energy.

Doubts and difficulties. It is true that fear urges to precipitate flight, rage to violent attack, and thus both emotions could be said to *"energize"* the organism. But this impulse to action is mediated over central pathways and is not necessarily the result of the simultaneous excitation of peripheral sympathetic pathways or the accompanying adrenaline secretion.

When immediate escape from danger is not possible, when danger continues and fear with it, there is a persistent desire to escape but no facilitation of any action that can be undertaken short of flight. On the contrary, there is a peculiar difficulty of acting which may become a downright inability to move or speak. In stage fright, for instance, thinking as well as speaking become more and more effortful, movements become forced, stiff, and ungainly. The many symptoms of sympathetic excitation seem to aggravate the situation: hands and feet are cold and trembling, the heart is racing, the tongue seems to stick to the roof of the mouth, the knees are so weak that it is difficult to stand or walk. The same incapacity to move or think is exemplified by pilot trainees who "freeze to the controls" on their first pilot flight.

This difficulty may occur even when there is a chance to escape, and may result in complete paralysis of action, for instance, in the moment just before an automobile collision when quick action might still avert it.

Psychologically speaking, in these cases fear has turned into despair: the appraisal that this must be escaped has turned into the appraisal that this is so overwhelming that it cannot be escaped. It is the feeling of powerlessness and incompetence which gives ground for such a change of appraisal, and this feeling of powerlessness seems to come with fear.

Animal experiments show the same reduction of activity during persistent fear. Hall (1941), for instance, found rats less active when exposed to the "open field," a large, open, well-lighted enclosure. These rats showed many signs of fear. They refused to touch the offered food and kept close to the walls. More recently, Tseng (1942) has confirmed this marked reduction of activity in fear situations, and Richter (1951) reported that wild rats reacted to a series of poisoning attempts either by ceasing to eat and starving to death, or by a marked and unusual immobility. The rats either sat, stood, or hung from the wire mesh of the cage, always in the same position, never moving or running around. Richter thought that this immobility was the result of "an ever-present fear of being poisoned." Mahl (1949) also found a reduction of activity in dogs when he gave them electric shocks daily for some time. They gradually became less active, slept less, and began to posture in odd ways. Arnold (1944) found this immobility in rats exposed to intense sound; it could be duplicated by the injection of adrenaline in dosages comparable to the secretion during fear.

Reduction of activity occurs not only in acute fear but also in chronic fear, as shown by Richter's rats and Mahl's dogs. It can apparently be transmitted from generation to generation. Hall's specially bred "emotional" strain of rats (which showed symptoms of fear rather than of other emotions) were both less active and less aggressive than normal rats. Neurocirculatory asthenia, which apparently is indistinguishable from anxiety neurosis, seems to have a high familial incidence and reduces the patient's activity (Cohen and White, 1951). Such patients have complained that they could not do much work or take normal physical exercise. These facts seem to indicate that fear does not always aid action but may hinder it if the danger continues and the fear is extreme or becomes chronic.

Cannon explained the harmful effect of chronic anxiety by pointing to the difference between a temporary emergency in which all the resources of the organism are thrown into the breach and long drawn-out emergency states that eventually exhaust these resources. This analogy implies that resources are withdrawn from the economy of the organism for defense in the same way as consumption is curtailed during war. However, in the economy of a country, the raw material needed for weapons really is withdrawn from home consumption; but in the organism, the accumulated "war matériel" can only be muscular energy or, more precisely, the assimilated food which provides it. As long as that is available, it can be used for any kind of muscular activity, peaceful or defensive. If it is true, as Cannon says, that sympathetic excitation and adrenaline secretion provide muscles with available glycogen, help in the utilization of blood sugar, and abolish the effects of muscular fatigue, this action should aid in muscular activity of every kind, not merely when muscles are used for flight. Either the sympathetic system and adrenaline facilitate the assimilation of energy-giving materials, as Cannon maintains (and then it is hard to explain why it should not do so continually); or they dissipate these materials (and then they will do so even in an emergency and cannot be said to "energize" the organism).

To decide between these two alternatives, we propose to examine the evidence that led Cannon to his concept of emergency action. But before we do so, we must distinguish between the central effects of fear and its peripheral effects.

FEAR AND EMERGENCY ACTION

In discussing the various action patterns observed in emotion, we have suggested that fear is an action tendency mediated by a special pathway within the general action circuit. When this pathway is activated, the experience and expression of fear with the urge to escape seem to occur. The electrical stimulation of this pathway induces violent action, which has led Hess (1949), for instance, to suggest that this region is an "ergotropic zone." Since adrenaline and noradrenaline seem to be synaptic transmitters in the hypothalamic link of the action circuit (see Vogt, 1954), an injection of either of these drugs should stimulate the action circuit.

We have attempted to show that this circuit sends relays to the motor cortex, which would suggest that its stimulation by adrenaline should facilitate muscular contractions. Several investigators have reported that large doses of adrenaline and noradrenaline lower the convulsion threshold and prolong convulsions (Hall, 1938; Keith and Stavraky, 1935; Minz and Domino, 1953). On the other hand, small doses of adrenaline seem to depress activity and suppress convulsions (Gellhorn *et al.*, 1939;

Arnold, 1944). It seems likely that large doses stimulate the central circuits before the peripheral effects of adrenaline have time to develop, while small doses are not sufficient for central stimulation. This inference is supported by Minz and Domino's finding that these hormones prolong convulsions even when adrenergic blocking agents have reversed the rise of blood pressure produced by adrenaline and reduced the rise of pressure caused by noradrenaline. Here the peripheral effects of adrenaline were prevented without changing its central effects.

Fear or adrenaline injection excites a pattern of relays in the action circuit which includes not only impulses to somatic motor nerves but also impulses to fibers in the pyramidal and extrapyramidal pathways that connect with vasomotor and other sympathetic nerves. This excitation of the sympathetic nervous system is intensified by the simultaneous secretion of adrenaline from the adrenal medulla (Cannon, 1915, 1932). It is this peripheral effect of fear or adrenaline which Cannon believed responsible for the energizing action of fear. Of necessity, this effect develops slowly and lasts for some time; it should increase and become cumulative if fear becomes chronic.

Rogoff and Stewart (1926) challenged Cannon's method of adrenaline determination in excited animals and claimed that the physiological changes during fear are the result of sympathetic excitation rather than adrenaline secretion. Since adrenaline is secreted when the sympathetic splanchnic nerves are excited, and later studies have invariably confirmed that the adrenaline content of the blood is increased during fear (Darrow and Gellhorn, 1939; Milhorat and Diethelm, 1947; and others), it can be taken for granted that fear induces both sympathetic excitation and adrenaline secretion, though it is possible that many of the effects Cannon ascribed to adrenaline are produced by the stimulation of sympathetic nerves (Celander, 1954). We now propose to show that the physiological effect of sympathetic stimulation and adrenaline secretion is such that it can account for the depression of activity found in chronic fear.

Physiological effects of adrenaline. Cannon's conclusion that adrenaline (and therefore fear) prepares the organism for fight or flight was based, first of all, on the earlier observation by many clinicians that the adrenal glands are necessary for muscular activity. If they are removed, there is a gradually increasing muscular weakness which finally ends in prostration and death. Cannon also referred to Oliver and Schäfer's results (1895) which showed that an extract from crushed adrenal glands increased and prolonged the contraction of normal resting skeletal muscle. Finally, Cannon himself found that adrenaline injection or perfusion restored the size of muscular contractions in fatigued nerve-muscle preparations. From these observations Cannon concluded that adrenaline secretion in the intact organism is necessary for proper muscular functioning, especially during emergencies (cf. Cannon, 1915).

When Cannon first proposed his theory, adrenaline was believed to be the only secretion produced by the adrenal glands. Since then, a great deal of research has established that the adrenal cortex also secretes hormones. In fact, it is the adrenal cortical hormones or some of their fractions that are essential for life and maintain muscular strength. If adrenal cortical extract is supplied, animals survive normally after removal of the adrenals though adrenaline secretion is now impossible.

Not suspecting that the adrenal cortex also secretes hormones, Cannon and his coworkers frequently used bilaterally adrenalectomized animals together with animals which had one adrenal removed and the other denervated. They did not always list the data for these animals separately, and so the deficits noticed after exclusion of the adrenal cortex may have been ascribed to the lack of adrenaline secretion. After the discovery of adrenal cortical hormones, research was almost exclusively concerned with these hormones without attempting to reevaluate conclusions from work in which adrenal medulla and adrenal cortex had been equated.

In a recent review, Ramey and Goldstein (1957) pointed out that the practice of Cannon and his coworkers to "treat as indistinguishable bilateral adrenalectomy and unilateral adrenalectomy, must indicate that the additional procedure of extensive sympathectomy is protective in nature." But on the whole, they accepted Cannon's conclusions concerning the effect of adrenaline secretion, without accounting for the contradiction. When the effect of adrenocortical secretion is taken into account, some of Cannon's conclusions may have to be modified. For instance, the ablation of one adrenal may impair muscular power temporarily until the adrenal cortex of the remaining gland enlarges and compensates for the loss. It makes a difference whether the temporary loss is ascribed to a deficit in adrenaline secretion or a reduction in the output of adrenal cortical hormones. For this reason, it seems advisable to examine the evidence in detail, at least where it is relevant for our purpose.

Adrenaline and muscular contractions. In their original articles, Oliver and Schäfer reported that they used extracts from the whole gland for their experiments, though in some of them they used extracts from the adrenal cortex and the medulla separately. When they injected the equivalent of 1 to 2 grains of fresh gland into a frog, they found a gradual arrest of all movement. When the muscle was electrically stimulated, it contracted; but the relaxation period after each contraction was greatly delayed. A larger dose had the same effect in a rabbit. These authors suggested that the adrenal gland (or at least the medulla) must have a secretory function (not an excretory function, as had been thought up to then), and that it was probably important for "maintaining the tonicity of the muscular tissues in general and especially of the heart and the arteries" (1895, p. xiii).

But delayed relaxation after contraction is not synonymous with improved muscular performance. The increase in tonicity may contribute to cumulative general muscular *tension,* both in smooth and striped muscles, but is not evidence that adrenaline or medullary extract improves phasic contraction. This "increase in tonicity" in the frog and rabbit was compatible with near-abolition of all reflexes and apparent paralysis. Such delayed relaxation might account for the catatonic immobility of Richter's rats and Mahl's dogs during chronic fear. We know that general muscular tension is higher in anxiety neurotics (Jacobson, 1934) though their work capacity is reduced.

Cannon's own work showed that the injection of adrenaline, either before or during work, does not increase work capacity, and adrenaline perfusion does not increase the contraction of rested skeletal muscle. Conversely, the bilateral removal of the adrenal medulla or of the sympathetic nervous system does not reduce work capacity in animals (Campos *et al.,* 1928; Cannon, 1932; Harris and Ingle, 1940). In fact, Harris and Ingle found that demedullated rats showed a slight but significant increase in performance. Campos and coworkers (1928) found that adrenal denervation on one side and removal of the other adrenal eventually produced better performance and longer endurance. According to their work curves (pp. 686, 688), exhaustion sets in earlier and work decreases after adrenaline injection—which corresponds to Harris and Ingle's results. Moreover, Brouha *et al.* (1936) found that performance was reduced for about six weeks after removal of one adrenal and demedullation of the other. This is about the time required for the growth of additional adrenocortical tissue to compensate for the loss of one adrenal.

In a later investigation, Ingle and Nezamis (1949) found that the work performance during faradic stimulation of the hind legs of adrenalectomized rats was proportional to the amount of adrenal cortical extract given intravenously. Injection of glucose markedly improved performance but adrenaline in small doses had no effect. In larger doses it suppressed work. Apparently, the injection of adrenaline does not prevent fatigue or increase performance in the intact animal; in fact, large doses decrease performance and increase fatigue. Conversely, the absence of adrenaline secretion or sympathetic stimulation does not impair muscular work; in fact, it may actually enhance it.

There is direct evidence also that adrenaline reduces muscular contractions. Gellhorn, Darrow, and Yesinick (1939) found that adrenaline in small doses diminishes or prevents insulin convulsions, even though the blood sugar remains low; it also prevents or reduces metrazol convulsions. Darrow and Gellhorn (1939) reported that adrenaline in physiological concentration reduces somatic hyperexcitability; it prevents sound-produced seizures in susceptible rats and also in rats made susceptible by strychnine injections (Arnold, 1945). Qualitatively, it was observed that adrenaline injection reduces muscular tension and results in flaccidity and sometimes in prostration, even when given in physiological amounts. It also abolishes or prevents strychnine convulsions (Arnold, 1942). Gellhorn (1953) suggested that such reduction of muscular contractions is a peripheral effect while adrenaline facilitation is a central effect. And Mohme-Lundholm (1953) has shown that this relaxing effect of adrenaline occurs because glycogen is broken down into lactic acid and is unavailable for muscular contractions. Drugs inhibiting such breakdown prevented not only the formation of lactic acid after adrenaline injection, but also the usual adrenaline inhibition. This breakdown of glycogen to lactic acid requires some time before it will inhibit muscular contractions; massive doses of adrenaline seem to exert their stimulating effect on central circuits before the peripheral effects have developed sufficiently to inhibit muscular activity.

We would suggest that central stimulation in fear produces increased muscular contractions, representing a "readiness to act" which is expressed as heightened muscular tension. Gradually, the peripheral effects of sympathetic stimulation in fear make it difficult for the muscles to respond with strong phasic contractions; consequently, there will be reduced work capacity yet increased muscular tension (e.g., in anxiety neurotics; see Jacobson, 1934). Excessive sympathetic stimulation (e.g., in terror) seems to make it impossible for the muscles to respond even with tonic contractions; instead, there will be flaccidity and prostration.

There are some findings, however, that have always been considered convincing evidence for an energizing effect of adrenaline: Cannon showed that adrenaline increases muscular contractions in the fatigued nerve-muscle preparation, and Campos and coworkers found that exhausted dogs began to work again on a treadmill if given small doses of adrenaline. It could be objected, of course, that these findings are of purely pharmacological interest. The normal organism could not secrete adrenaline when fatigued because intense exercise completely exhausts the adrenal medulla (Cameron, 1936). Nor could fear and sympathetic excitation occur at this point, because the readiness to react emotionally is severely curtailed in exhaustion. This is illustrated by the feeble response of rats to teasing after a sound-produced seizure, and also by Campos *et al.'s* report that they could not arouse "excitement" in exhausted dogs.

Though the renewed capacity to work after adrenaline injection cannot be used to prove that fear or adrenaline energizes the rested organism, the sheer fact requires an explanation. We have suggested that the injection of adrenaline activates the central circuit mediating fear, and will induce increased muscular activity. In complete exhaustion, when the adrenal medulla is depleted, a moderate dose of adrenaline might be expected to activate this circuit without stimulating the sympathetic nervous system sufficiently to depress muscular contractions. If, on the other hand, a large dose is given, enough adrenaline will be present to produce violent excitement and eventually peripheral symptoms. According to Campos and associates, a large dose of adrenaline (from 0.04 milligram per kilogram up) injected into exhausted dogs produced excitement, increased fatigue, and prevented further work.

The recovery of contraction in the fatigued nerve-muscle preparation after adrenaline injection or perfusion can be explained by the fact that adrenaline in weak concentration augments the effect of acetylcholine, and so improves transmission across the myoneural junction. Luco (1939), for instance, reported that small doses of adrenaline produce a fleeting depression of a fatigued muscle stimulated through its nerve, which is followed by a secondary augmentation. Since large doses of adrenaline produced only depression, and this depression was prevented by ergotoxin (which blocks sympathetic effects), the depression seems to be the primary (adrenergic) effect, while the secondary augmentation seems to result from improved cholinergic transmission. Bülbring and Burn (1940) reported similar findings and showed that this secondary augmentation of muscle contractions after adrenaline was accompanied by a fall in blood pressure, indicating that sympathetic stimulation had ceased.

In a careful review, Burn (1945) showed that adrenaline in weak concentration increases the action of acetylcholine, both in the central nervous system and in the ganglia of the sympathetic system. In strong concentration, cholinergic transmission is depressed. In other words, the improved muscular contractions after small doses of adrenaline are the result of a transmission of cholinergic impulses which is activated or potentiated by adrenaline. When too much adrenaline is present, muscular contractions are inhibited. Burn suggested that the paralyzing effect of fear may be the result of increased adrenaline secretion which inhibits muscular activity.

Cannon discovered that transmission in sympathetic postganglionic nerves is mediated by two adrenaline-like substances which he called "Sympathin E" and "Sympathin I." The former excites, the latter inhibits. Later research has shown that these two kinds of neurohumors roughly correspond to two different hormones secreted by the adrenal medulla but also found in the brain and in sympathetic ganglia: noradrenaline and adrenaline. Of the two, only adrenaline potentiates acetylcholine action, but both hormones seem to stimulate the action circuit. Adrenaline was secreted by the adrenal medulla when points in the hypothalamus were stimulated which induce the "flight" pattern; and noradrenaline was secreted on stimulation of an area from which Hess has reported "rage" reactions (cf. Folkow and Von Euler, 1954).

Now let us turn to the mechanism by which adrenaline or sympathetic nervous system stimulation was thought to help put the body on an emergency footing.

Adrenaline and the circulation. Cannon held that adrenaline produces vasoconstriction in the viscera and in the blood vessels of the skin, but vasodilatation in skeletal muscles. Thus adrenaline secreted during emotional excitement would drain the blood from the viscera where it is not needed and shift it to the limbs where it is.

We have seen (Ch. 3) that the initiation of movement is accompanied by a dilatation of blood vessels in the active muscles which is mediated over sympathetic *cholinergic* nerves. This is a central effect that occurs with any action, long before the adrenaline secreted during fear could begin to act. In the resting muscle, adrenaline decreases the blood flow (Gellhorn, 1943). With larger dosage, adrenaline can produce vasoconstriction even in active muscles (Luco, 1939). Injection of acetylcholine into the mesenteric artery produces extreme dilatation, while the injection of small doses of adrenaline produces an initial dilatation (because it potentiates acetylcholine) and later constriction; but larger doses of adrenaline, or the injection of noradrenaline, results in constriction (Celander, 1954). Thus the primary effect of both adrenaline and noradrenaline seems to be vasoconstriction.

The effect of sympathetic stimulation or adrenaline on blood flow in skeletal muscle is still controversial. But the view that such stimulation increases the blood flow seems to be based on inference from Cannon's theory as much as on actual evidence. For instance, it is held that the reduced blood flow in the hand during fear and pain is the result of vasoconstriction in the skin which masks the dilatation of blood vessels in the muscles (Selye, 1950, p. 504). But adrenaline, noradrenaline, and sympathetic stimulation produce vasoconstriction even in the skinned limb (Celander, 1954), and blood flow in the resting forearm is increased after sympathectomy or blocking of sympathetic nerves. Adrenaline injected into an artery in doses large enough to increase the blood pressure also reduces the blood flow in the forearm (Harpuder *et al.*, 1947).

The rate of blood flow depends primarily on the force with which the heart pumps blood into the arteries. When the arteries are constricted, the blood pressure will rise correspondingly, provided the heart action remains constant. But during sympathetic stimulation, the rise in blood pressure does not necessarily parallel the constriction of blood vessels because the heart muscle probably cannot maintain an unchanged cardiac output (Celander, 1954). In addition, it is known that both adrenaline and noradrenaline impair the capillary walls so that fluid and proteins are lost to the surrounding tissues (Gellhorn, 1943; Ramey and Goldstein, 1957); the peripheral resistance drops and blood flow decreases. This is confirmed by Freeman and coworkers (1941) who found that the continuous intravenous injection of adrenaline (0.0034 to 0.1164 milligrams per kilogram per minute) in dogs resulted in increased blood pressure and decreased rate of blood flow. The mucous membranes gradually became pale until limbs and ears were cold and had a doughy feel, the pulse was thready, the blood flow in the ear veins was very slow, the blood dark, and the dogs eventually died in shock. If this is the result of an infusion not greatly exceeding the adrenaline secreted during fear,* we cannot assume that intense and long-continued fear will have an energizing effect. Far from having beneficial effects in surgical shock, sympathetic stimulation or adrenaline infusion can produce shock. Total sympathectomy, on the other hand, *prevents* shock from hemorrhage in dogs (Freeman *et al.*, 1938), as does procaine block of the adrenal nerves in man (Parkins *et al.*, 1941). Neither the sympathetic nervous system nor adrenaline counteracts the effects of hemorrhage, as Cannon assumed, but seems to aggravate them.

Adrenaline and the heart rate. Cannon also suggested that the increase in heart rate and blood pressure produced by adrenaline materially aids in preparing the organism for emergency action.

Whether increased blood pressure and heart rate is favorable for muscular activity depends on the amount of blood available, its distribution in the body, and the force of the heart beat. If the amount of blood discharged at each beat (the stroke volume) remains constant, a faster beat will increase the output of the heart and improve the blood flow. When the heart rate increases, the stroke volume will also increase if the blood returns to the heart through the veins in sufficient volume and at sufficient speed. But the volume of the venous blood decreases in emotion because of seepage through the capillaries. Unlike exercise and physical work, where the active muscles help to speed the return of venous blood, emotion that stops short of action does not improve venous return.

According to Cannon's own findings (1932, p. 152), the excitement of a cat facing a barking dog accelerated the denervated heart from 120 to 160 beats per minute, as long as the adrenals could be stimulated through the sympathetic nervous system. When the connection was interrupted by denervating the adrenals, emotional excitement only increased the heart rate from 118 to 120 beats per minute. But according to Cannon's graph, the recorded amplitude of the heart beat was doubled after the adrenals were denervated. In this case, the stroke volume apparently did not remain constant during excitement, and the output of the heart was probably reduced.

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* In dogs whose sympathetic nervous system has been blocked by complete spinal anesthesia, normal blood pressure can be maintained by a continuous infusion of adrenaline at the rate of 0.45μg/kg/min; consequently, the total amount of adrenaline active in the body has been calculated as 0.71μg/kg (Guyton and Gillespie, 1951). According to Bülbring and Burn (1949), 20 to 80 percent of the effect of adrenal blood collected during stimulation of the splanchnic (sympathetic) nerves is due to noradrenaline. And stimulation of the splanchnics provokes a discharge of as much as 100μg of adrenaline (Bülbring and associates, 1948); thus an infusion of 3 to 116μg, as in Freeman's experiment, may not be too far above the physiological limit.

In man also, an increase in heart rate does not always mean an increase in blood flow. With a heart rate of more than 100 beats per minute, there is a decrease in stroke volume and the heart actually reduces its output. Such a rate is not unusual during fear. Duncan and others (1951) reported, for instance, that patients with anxiety neurosis had a decreased cardiac output during the fear and apprehension connected with their first visit to the clinic. In one case, the heart rate increased to 128 beats per minute, yet stroke volume and cardiac output decreased appreciably.

Characteristically, the gradual improvement in efficiency during athletic training is accompanied by a *decrease* in the heart rate both at rest and during exercise (Borgard, 1937). According to Borgard, strenuous exercise results in a simultaneous increase of heart rate, stroke volume, and oxygen utilization; but the heart rate of athletes increases much less than that of untrained men and the stroke volume increases far more. Indeed, some athletes show no increase in heart rate during moderate exercise, but a considerable increase in stroke volume which takes care of the increased oxygen requirement (Best and Taylor, 1951). Consequently, work capacity does not depend exclusively or even primarily on the increased heart rate. This is confirmed by the study of Duncan *et al.* which showed that blood pressure and heart rate decreased in patients with anxiety neurosis after successful psychotherapy while their exercise tolerance increased.

Adrenaline and blood sugar. Cannon further suggested that adrenaline energizes because it provides the muscles with an abundance of fuel by releasing stored glycogen from the liver.

However, adrenaline releases glycogen not only from the liver but from the muscles as well. Major and Mann (1932) have shown that adrenaline infusions decrease the glycogen content of muscles even when the concentration is so low that there is no rise in blood pressure (0.00016 milligram per kilogram per minute). Larger doses cause a more marked decrease in muscle glycogen. Since fear increases the blood pressure markedly, we may assume that the adrenaline secreted during fear will significantly reduce muscle glycogen.

It seems generally accepted today that adrenaline acts on sugar metabolism by breaking down muscle glycogen to lactic acid (glycolysis) and releasing glycogen from the liver (glycogenolysis). It does not contribute to the assimilation of blood glucose by muscle or other tissues; on the contrary, adrenaline seems to inhibit glucose utilization by slowing down its transfer from body fluids to tissue cells (Somogyi, 1951). For the *formation* of glycogen in liver and muscle, the carbohydrate hormones of the adrenal cortex (glucocorticoids) and insulin are necessary, but adrenaline has no part in this action (Verzár, 1939; Britton and Corey, 1940).

From this evidence we are forced to conclude that the increased blood sugar released by adrenaline secretion is not immediately available to the muscles; in fact, the presence of adrenaline seems to prevent glucose utilization. But it may be objected that adrenaline at least helps to convert lactic acid into glycogen indirectly, by increasing the respiratory rate, thus supplying an abundance of necessary oxygen. Let us see whether that is the case.

Adrenaline and oxygen utilization. When the muscle contracts, glycogen is converted to lactic acid. If enough oxygen is available, the acid is immediately reconverted to glycogen, and there is practically no accumulation of lactic acid in blood or tissues. When oxygen is lacking or its supply inadequate, lactic acid accumulates. Its resynthesis into glycogen is first slowed and then prevented until further muscular contractions become impossible. Since adrenaline increases the respiratory rate, Cannon suggested that this is accompanied by an increased supply of oxygen which helps the laboring muscles by reconverting lactic acid to glucose.

However, adrenaline increases lactic acid concentration even in the resting organism. The increased oxygen consumption after adrenaline injection (20 to 40 percent, according to Best and Taylor, 1951) is needed for the resynthesis of lactic acid into muscle glycogen. During strenuous exercise, there is more demand for oxygen than can be provided, even when the respiratory rate increases maximally. As work continues, muscle glycogen is synthesized at a slower and slower rate until no more is available for muscular action, and man or animal is exhausted.

In fact, adrenaline alone and strenuous work alone will exhaust muscle glycogen and increased oxygen will be needed to convert lactic acid back to blood glucose and muscle glycogen. If these changes are in the same direction so that either adrenaline alone or exercise alone eventually exhausts muscle glycogen, how can adrenaline (or sympathetic stimulation) provide additional energy reserves? Far from so doing, adrenaline draws upon the same glycogen store that is needed for muscular contraction. Thus it becomes understandable why fear or an adrenaline injection gradually inhibits muscular contractions and sometimes results in complete prostration.

There are some studies that have examined the functioning of the bodily economy during strenuous work. These should either prove or disprove our interpretation.

Muscular efficiency. Strenuous athletic training for six months has produced several changes in the bodily economy which seem to imply that sympathetic activity has been reduced. Men in training who found a run exhausting at the beginning of training, found it well within their capacity at the end; at that time, such a run resulted in considerably lower blood sugar and blood lactate levels than at the beginning (Robinson and Harmon, 1941). Since the oxygen requirements for the run remained the same, the breakdown of glycogen to lactic acid and its resynthesis into glycogen must have been more efficient, probably because adrenaline secretion was reduced. Von Euler (1953) pointed out that running per se does not result in increased adrenaline secretion. Such an increase is noted only when exhaustion sets in.

Patients who suffer from anxiety neurosis (variously called effort syndrome, vasomotor neurosis, neurocirculatory asthenia) do not tolerate exercise well. They show early exhaustion, increased lactic acid concentration, and greatly augmented oxygen consumption even during mild exercise. There is no doubt that these are effects of increased sympathetic stimulation with increased adrenaline secretion during chronic anxiety. During fear and tension, normal people have a similarly lowered exercise tolerance with symptoms of sympathetic overactivity (Duncan *et al.*, 1951).

Cohen and White (1951) have summarized a number of studies that throw light on the mechanism by which fear and anxiety impair muscular activity. These studies show that normal people could perform an exhausting treadmill run for twice as long as could patients with acute anxiety. The latter patients in turn showed twice as much endurance as did patients with chronic anxiety states. Apparently, the harmful effects of anxiety become more pronounced as time goes on. This is not surprising if fear or anxiety are cumulative, as we maintain.

Even during moderate exercise, the oxygen uptake of the neurotic patients was lower than that of their normal fellows, despite the fact that their faster breathing supplied them with more air than did the lower respiratory rate of normals. Both lactic acid and blood sugar of neurotics were higher than was found in the normal group even during moderate exercise (treadmill walk). During exhausting exercise (treadmill run), neurotics had a much lower oxygen consumption than normals and their blood lactate was three times higher. This means that the increased sympathetic stimulation in anxiety raises the respiratory rate during exercise but does not increase oxygen consumption. Lactic acid accumulates quickly, and very soon there is no more glycogen for muscular contractions and fatigue sets in.

This long look at the physiology of adrenaline shows that the subjective experience of weakness during acute or chronic fear is supported and explained by objective evidence. Adrenaline and sympathetic stimulation do not improve muscular performance. Rather, they reduce efficiency by increasing lactic acid formation and by interfering with glucose and oxygen utilization. Sudden fear may bring a sudden urge to flee and so provide a powerful spur to action. When this urge leads to successful escape and the danger is past, the effects of sympathetic stimulation quickly subside. But when escape is impossible and fear becomes chronic, the physical and psychological effects of fear soon incapacitate a man for serious work. Mental work becomes impossible because attention is centered on the threatening danger. The central effect of fear (see Ch. 5, Drug Effects) makes it difficult to remember, imagine, or decide on action. Physical work becomes increasingly laborious because the cumulative effect of sympathetic stimulation seriously reduces muscular efficiency.

ANGER AND EMERGENCY FUNCTION

According to Cannon's emergency theory, anger as well as fear induces sympathetic excitation and adrenaline secretion. But psychologically speaking, anger is different from fear. It is based on a different appraisal, namely, that something harmful can be overcome, rather than the appraisal that this something is so overwhelming that it is difficult to escape. Also, anger is experienced differently, as a tendency to fight, strike, and tear, instead of an urge to cower or escape. There is a sensation of tension and fullness instead of the sensation of weakness and tremor so noticeable in fear. Considering that the appraisal and the psychological experience are different, that the proposed action is different, and that the bodily sensations differ as well, there must surely be differences in the pattern of central nervous excitation and therefore in the physiological state. We have sketched the central action pattern in Chapter 6. What remains is to review such evidence as we can find that will throw light on the physiological state.

Anger and gastric function. Perhaps the most convincing evidence for a physiological difference between anger and fear is found in the admirable studies of Wolf and Wolff (1943) on human gastric function. These authors describe careful observations over a number of years on "Tom," a man with a chronic stomach fistula, in whom the blood flow to the stomach lining as well as the activity of the stomach could be observed in many different situations.

Wolf and Wolff found that fear regularly reduced gastric activity and blood flow. The situations they described doubtlessly did arouse fear. On one occasion when Tom's fistula was examined, a doctor came into the room, very annoyed because he had been looking for a record that was unaccountably missing. Tom had mislaid it but did not say anything because he was afraid he would be dismissed if his negligence were discovered. On that occasion, his stomach was practically bloodless and completely flaccid.

Whenever Tom was annoyed, angry, or resentful, his stomach reddened, and at the same time, stomach contractions and acidity increased. Another patient with a fistula showed similar changes on occasions when he became angry and used profane language. After the stomach was denervated by cutting the vagus nerve, this patient still had a flushed face and used profane language when he became angry, but his stomach no longer showed any changes. Wolf and Wolff also reported lowering of blood pressure (a cholinergic symptom) in a patient with hypertension whenever he was outspoken in his resentment of his wife, but a rise when he was presented at a conference and felt apprehensive.

During the last few years, several vagotomies have been performed on patients with gastric ulcer who did not improve on diet and drugs. Before the operation, ulcer pain was caused or aggravated by various emotional upsets, by anger, hostility, resentment, and worry. After the vagus was cut, such emotions no longer had any effect on their well-being. Parasympathetic excitation induced by these emotions could now no longer increase gastric acidity or stomach contractions, and could not aggravate the ulcer pain.

These observations demonstrate beyond any doubt that anger produces effects on the circulation that are different from those produced by fear. Wolff (1950) has listed an impressive number of similar changes seen in the lining of nose, colon, and vagina, all of which seem to indicate that anger and resentment result in cholinergic excitation, while fear and anxiety seem to excite adrenergic nerves.

Anger and excretory functions. Emotional defecation is abundant during fear (Hall, 1941; Tseng, 1942; Fleetwood and Diethelm, 1951) and ceases during anger (Tseng, 1942). Fleetwood and Diethelm mention it as one of the symptoms of marked anxiety, but do not list it as a symptom of anger or resentment. When parasympathetic influence is excluded, as for instance in vagotomy, there is often persistent diarrhea.

Tears, abundant during temper tantrums, are completely inhibited during fear—and the secretion of tears is the effect of parasympathetic excitation (Lund, 1930). Frequency of urination, which occurs both in fear and anger (Tseng, 1942) may be caused by two different mechanisms: during fear, by the relaxation of the sympathetically innervated sphincter; during anger, as a result of bladder contraction (a cholinergic symptom), which induces a reflex relaxation of the sphincter.

Cholinergic hormones in anger and resentment. Adrenaline injection has induced anxiety attacks in neurotic patients, but so has the cholinergic drug mecholyl (Lindemann and Finesinger, 1938; Funkenstein and associates, 1949). However, the attacks differed according to the drug that produced them. Adrenaline injection was followed by pallor, tremor, weakness, and great fear; but mecholyl induced an attack with flushing, sweating, an increased flow of saliva, and marked agitation. These surely are two different if not actually opposed patterns, the first best described as fear, the second as agitation; and agitation is akin to worry rather than simple fear. We have argued in the preceding volume (Ch. 12) that worry implies not a withdrawal from danger, as does fear, but a desperate urge to *do* something to cope with the danger. This includes ceaseless mental activity which gives rise to the characteristic agitation and may account for the cholinergic effect.

It may well be that the cholinergic excitation in agitated worry is accompanied by a secretion of noradrenaline. Fleetwood and Diethelm (1951), for instance, found a substance in the blood of patients suffering from "anxiety" which was tentatively identified as noradrenaline. Other investigators reported that an injection of noradrenaline does not produce anxiety while adrenaline does (Smith, 1951; and others). This discrepancy seems to hinge on the meaning of the term "anxiety." Fleetwood and Diethelm's patients may have suffered from agitated worry (mecholyl anxiety), while Smith's patients may have felt acute fear (adrenaline anxiety).

Fleetwood and Diethelm also found evidence for several kinds of cholinergic stimulation during states of tension and anger. For instance, they found a cholinergic substance in human blood during emotional tension states (characterized by irritability, muscular tension, insomnia, inability to concentrate). Another cholinergic substance was found during anger and resentment (characterized by desire for revenge, hostility, sarcasm, criticism of others). It is tempting to suggest that the emotional tension might be a chronic form of anger or resentment, particularly because the cholinergic activity of the "tension" substance was found to be considerably stronger than that of the "resentment" substance. Fleetwood and Diethelm insisted, however, that these substances are not identical, though they both have a cholinergic action. Apparently, both tension and resentment were relieved by alcohol, while anxiety was reduced but not abolished. According to Smith (1951), the anxiety, tremor, and nervousness produced by an adrenaline injection were abolished if a drink of whisky was given at the same time. His patients commented that the adrenaline effect felt like the "shakes" they had during a hangover, which also were abolished by taking a drink. We would suggest that alcohol depresses both the "anger" and the "fear" pattern in the action circuit, but apparently affects acetylcholine transmission more than adrenergic transmission, and among adrenergic nerves, affects noradrenaline-sensitive nerves more than those that are stimulated by adrenaline.

Noradrenaline and anger. Funkenstein *et al.* (1952) suggested that a profound fall of blood pressure after the injection of mecholyl indicates excessive secretion of an adrenaline-like substance, while a moderate fall indicates excessive secretion of a noradrenaline-like substance. They based this inference on the observation that people given adrenaline show a significant fall of blood pressure after mecholyl while those given noradrenaline show a moderate or slight fall.

In a frustration experiment, Funkenstein *et al.* (1953) asked young men to solve difficult problems without paper and pencil, blaming them for stupidity when their answers were incorrect. Some of the men showed outright anger at this treatment ("anger out") while others blamed themselves or apologized ("anger in"). Still others showed anxiety. Funkenstein and associates found that individuals who showed "anger out" had a cardiovascular pattern similar to that found after noradrenaline injection, that those who showed "anger in" had an adrenaline pattern, and that those who showed anxiety had an exaggerated adrenaline pattern. Psychologically speaking, self-blame ("anger in") is closer to fear than to anger because the person appraises the situation as difficult and himself as inadequate in dealing with it. He feels helpless though he recognizes that the situation is not really threatening, and becomes impatient with his own reaction. Another study (Ax, 1953) showed that anxiety or fear (the subjects were intensely afraid of a possible shock from defective electrical wiring) produces a cardiovascular pattern like that seen after adrenaline injection, while anger produces a pattern like that appearing after noradrenaline injection.

Vrij and associates (1956) reported that noradrenaline, like adrenaline, induces the release of glucose from the liver; but unlike adrenaline, it leaves muscle glycogen untouched. This would mean that noradrenaline secretion increases the blood sugar available to the muscles without breaking down muscle glycogen, and so promotes muscular strength. This would explain why anger gives us the feeling of being stronger than usual, in contrast to fear which makes us feel weak.

These findings justify the inference that anger is accompanied by noradrenaline secretion and consequent rise in blood pressure and by cholinergic vasodilatation. Accordingly, there should be a different pattern of sympathetic excitation. Folkow and Von Euler (1954) have shown that hypothalamic stimulation from some points induces adrenaline secretion and from other points, noradrenaline secretion. We have suggested that the "fear" pattern activates the former, producing vasoconstriction and adrenaline secretion, while the "anger" pattern activates the latter, inhibiting the vasoconstrictor fibers and inducing noradrenaline secretion.

That aggression (hence anger) and noradrenaline secretion go together is shown by the fact that animals that prey upon others, and attack and fight a great deal, have mostly noradrenaline in their adrenal medulla (the lion's medulla contains 60 percent noradrenaline against 40 percent adrenaline), while rodents and other plant eaters that need not attack other animals for food, but flee from predatory enemies, have more adrenaline than noradrenaline in their adrenal medulla (rabbit and guinea pig have 85 to 100 percent adrenaline). The same ratio holds within a species: the more fearful dogs secrete predominantly adrenaline, the more aggressive dogs predominantly noradrenaline (Funkenstein *et al.*, 1953).

Finally, anger affects the muscular system differently from fear. It has been found to raise the level of general activity markedly in rats (Tseng, 1942), while fear reduces it. Anger and fighting result in an enlargement of the adrenal cortex (Christian, 1956), which promotes muscular power. Indeed, the fact that anger or rage is accompanied by increased activity and increased strength is so well known that psychoanalysis makes aggression the prototype of all nonsexual activity.

However, extreme anger will lead to incoordination. The urge to violent attack does not favor finely coordinated action. Also, there is a point at which something inimical may be judged as so threatening that it will arouse desperation rather than anger. Or the expression of anger itself may produce fear: the other person may retaliate, or the attack may destroy something a man may not care to lose. If fear is aroused in addition to anger, it will intensify sympathetic excitation and may change the outward symptoms. Popular speech correctly has it that a man is "red with anger" and "hot under the collar," but also that he is "white with anger" and "trembling with rage."

Anger, panic, and startle. Anger seems to be related to other reactions that show heightened activity, such as startle, panic states and epileptic seizures, and also to the sound-produced seizures (sometimes called "experimental neuroses") in rats.

Tseng (1942), for instance, found that violent anger could be aroused in rats if they were confined in a body-size cage and teased with straws. After two weeks of daily teasing, ten out of nineteen rats had running seizures on sound stimulation. Of a comparable group of sixteen rats, left in their living cages undisturbed during these two weeks, only one had such an attack when retested. Since none of the total group had been found to have seizures on the initial test, it would seem that the difference in susceptibility between the two groups can be ascribed to the teasing of the experimental group which aroused vicious anger.

In an earlier publication (Arnold, 1944) it was suggested that the sound-produced seizure is an analogue to human epileptoid seizures rather than to human neurosis. This seems the more likely as it is now recognized that sound-produced seizures occur because of an inflammation of the middle ear which makes the rats more sensitive to noise. In epilepsy, there is increased susceptibility to stimulation (see Ch. 5), and thus intense sound or light may induce attacks.

It is a well-known fact that epileptic patients are inclined to be irritable and to have temper tantrums, and that anger or excitement often ushers in an attack. There is a similar connection between anger and epileptoid reactions (panic states) in traumatic war neuroses, as pointed out by Kardiner: "Easily aroused to anger, these patients are very prone to motor expression. They either break or tear objects in these fits of temper or strike the people who happen to be around them . . . If the outburst is accompanied by loss of consciousness, the patient is usually dangerous" (1941, p. 95). There is an obvious connection between such fits of temper and the psychomotor attacks we have discussed before (Ch. 4). We would suggest that in all these cases there is a hyperexcitability of the hippocampus, either constitutional (idiopathic epilepsy) or the result of traumatic experience (traumatic war neuroses) or, finally, acquired by a habitual indulgence in anger. In the third case, it seems to require an additional factor (like middle ear inflammation in sound-produced seizures) to lead to an epileptoid seizure.

Rats that have seizures on sound stimulation also show increased startle, and so did Kardiner's patients. This seems merely an indication that the action circuit is hyperexcitable.

OTHER EMOTIONS

Cobb (1950) thinks that love is accompanied by cholinergic excitation. He lists these symptoms: relaxed muscles, soft voice, vasodilatation, wide pupils, relaxation of intestines, rapid respiration, rapid heart beat, secretion of sex hormones, moist conjunctiva, salivation, and a feeling of warmth. Cobb emphasizes that these symptoms are characteristic for love and affection, not for sexual passion. However, this excitation would have to be moderate.

Psychologically, affection and sexual love are easily distinguished by their object and their aim. While all love aims to be with the beloved, the way of being together may range from liking to talk together, drink together, play together, to the love that wants to be together in the most intimate way possible. What is aimed for will depend on the appraisal of the person loved; and the emotion aroused by this appraisal will be mediated over the action circuit but show a different pattern in each case.

Only when there is a sexual attraction and a sexual aim will there be excitation of the sexual organs, and thus sexual passion. Only a nursing mother will have prolactin secretion when she sees her babe or even thinks of him; and only if she enjoys nursing will there be a "let-down" of milk during nursing (see Selye, 1934). There is evidence for saying that sexual love is a particular kind of love with a characteristic action pattern. To say that all love is basically sexual love and so imply that every action pattern that deviates from this "basic" one is aim-inhibited, smacks of theory without evidence. Some parts of the action pattern are similar in sexual love and in other kinds of love, but other links in the pattern are decidedly different. The physiological changes aroused will depend on the kind of love felt, and the emotional experience, in turn, will depend on the way in which this particular situation is appraised—as long as we remember that such an appraisal is both immediate and intuitive, but also reflective, at least in the human being.

Any emotion felt at a particular moment necessarily depends upon the appraisal of the specific situation. This appraisal may give rise to nuances and combinations of emotion that beggar description or classification. It is neither possible nor profitable to distinguish these emotions on the basis of their physiological pattern of autonomic changes or emotional expression. A careful analysis of the situation can give us a hint, however, of what type of emotion we are dealing with. Once we know the broad divisions, it will be possible to predict the probable effects of all emotions that are related to these broad types.

As a start, it is sufficient to know, for instance, that fear excites the sympathetic nervous system and induces adrenaline secretion which has undesirable effects on the body when long continued. Such knowledge will let us understand the effects of chronic fear in neurosis and will enable us to introduce effective countermeasures. We also know that the most favorable condition for activity seems to be produced by mild cholinergic excitation as aroused by affection and interest. Both fear and anger are a spur to action but lose this effect if action does not overcome the obstacle or does not take us out of danger. Continuing fear and anger reduce the efficiency of action and disturb the economy of the organism.

To consider excitement an emotion, as has been often done (Cobb, 1950; Arnold, 1945), does not seem feasible. Excitement is common to many emotions: worry, despair, joy, or anticipation. It may be pleasant or unpleasant. And it may or may not be present in any one emotion; joy may be excited or calm; disappointment, agitated or resigned. Thus excitement seems to be a factor in the action pattern that goes with emotion, and not an emotion in its own right.

There may be important connections between a given set of physical symptoms and a particular complex emotion. But what little we know of such relations owes more to popular wisdom and the efforts of novelists and poets than it does to experimental investigation. Homesickness, for instance, or longing for a particular person or thing, has long been known to have physical effects, as the word "home*sickness*" indicates. To guess at these effects, we must consider the kind of emotion to which homesickness might be related: sadness and grief at the absence of what we love; hope alternating with despair, depending on our confidence at a given moment that we may go back or the suspicion that we shall never do so. The investigation of such emotions would require both careful psychological analysis and ingenious methods, "psychosomatic" research of a high order. At the present time, psychosomatic methods are mainly employed to test inferences from speculative hypotheses rather than to explore the connection between emotional experience and physical effects.

PHYSIOLOGICAL CHANGES IN FEELING

Feelings, like emotions, are affective states that can arouse an impulse to action together with physiological changes. Earlier theorists have sometimes attempted to account for the experience of feelings as well as of emotions as a result of such physical symptoms.

F. H. Allport (1924), for instance, suggested that parasympathetic stimulation, supplemented by central nervous system effects, induces bodily changes that are felt as *pleasant* emotions or feelings; while sympathetic excitation induces bodily changes that are experienced as *unpleasant* emotions or feelings. Pain, fear, and rage, for instance, would result from changes brought about by the sympathetic nervous system; the pleasure of eating and sexual activity, from changes brought about by the parasympathetic system. According to Allport, some states of pleasantness, like the pleasure felt in games or bodily exercise, in elation and mirth, are the result of unimpeded conduction in somatic nerves.

We have mentioned before that the fibers conducting sensory and motor impulses cannot themselves carry the information that conduction is impeded or unimpeded. Sensory nerves can only mediate sense qualities, motor nerves can only mediate movement. If feeling is the result of conduction in somatic nerves, it must be mediated by structures that are different from those nerves; we have given evidence showing that the peripheral and central connections of the estimative system can indeed mediate an estimate of the effects either of sensation or of movement.

We would agree with Allport that pleasure is associated with parasympathetic and pain with sympathetic stimulation. But the autonomic nervous system can only be excited via some reflex connection with the sensory system. It is likely that local pain, like cold, can induce reflex vasoconstriction, a sympathetic symptom; but this reflex occurs, we think, because the effect of excessive stimulation, like the effect of cold, is gauged via the peripheral estimative fibers and induces a reflex contraction of smooth or even striped muscles (e.g., in appendicitis). These contractions do not cause the feeling of pain but may intensify it. This accounts for the fact that pain can be reduced (though not abolished) by muscular relaxation (see Hardy *et al.,* 1952). The feeling of pain is experienced, we believe, when impulses conducted over fine fibers both from somatosensory and sympathetic nerves reach the thalamic estimative nuclei and the cortical estimative area.

There is comparatively little experimental evidence that would allow us to connect specific physiological changes with feelings of pleasantness and unpleasantness, pleasure and pain. The vast literature on pain yields little information because the "sensation" of pain, the "suffering" connected with it, and the physiological changes have usually been treated as a combined "pain response" (see Barber, 1959). We have shown that pleasure and pain are feelings, specific reactions to the effect of somesthetic impressions, while pleasantness and unpleasantness are feeling reactions to sensory impressions from all modalities. Clearly, a man who feels pain also appraises the source of this pain, be it a blow, a disease, or even a laboratory experiment. He may feel anger, fear, resentment, or amusement, depending on the intensity of the pain and the meaning each of these situations carries for him. What is called "suffering" is the emotion of fear or anguish that follows his appraisal that the pain indicates a serious danger, and accounts for much of the difficulty encountered in the treatment of pain. The physiological changes seem to be partly the result of the feeling of pain and partly of the emotion evoked by the total situation.

For instance, Hardy and associates (1952) report that pain induced by tightening thumb screws roused one man to resent this procedure vociferously; his colonic mucosa became pale and bloodless. Another man calmly accepted the pain and his colonic mucous membrane remained normal. In another experiment, the subject's blood pressure rose on pain stimulation when he was afraid, but remained almost unchanged after his fear had abated. A woman showed a fall of blood pressure on painful stimulation when she felt hopeless and depressed, and a rise when she felt cheerful and confident. Such discrepancies clearly show that the physiological changes depend not only on the feeling of pain (i.e., the immediate and intuitive appraisal of excessive somatic sensations mediated by the peripheral estimative system) but also on the appraisal of the total situation that has brought it about.

Progress in this field can be expected only if these two factors are carefully controlled. This requires in addition to the usual experimental controls an account of the person's appraisal of the situation and a report of his feeling state.

8. HOMEOSTATIC REGULATIONS

The conception that the excitation of the sympathetic nervous system together with adrenaline secretion is an emergency reaction led Cannon to trace the interlocking mechanisms by which the organism maintains a dynamic equilibrium despite environmental changes. His theory of homeostasis is an ingenious extension of Claude Bernard's notion of the "milieu interne." For Cannon, it is the sympathetic nervous system that counteracts every kind of environmental pressure and the parasympathetic system that builds up bodily resources. The various bodily changes during emotion, as well as the reactions to heat and cold, are brought about by the sympathetic nervous system, according to Cannon, and act as homeostatic mechanisms.

FACTORS IN HOMEOSTASIS

In our context, the various factors that have to be explained by a theory of homeostasis merit careful discussion. Is emotion, like other organismic reactions, homeostatic and adaptive? Is it a survival reaction?

Emotion and homeostasis. In emotion, we are dealing with a psychological experience that follows upon perception and evaluation. As an action tendency, it is mediated by a pattern of motor impulses that are relayed to sympathetic or parasympathetic nerves, depending on the kind of emotion. Neither the emotional action tendency nor the action to which it urges is homeostatic. In the pursuit of his prey, the lion expends energy and incurs an energy debt. After he has caught and devoured his prey, metabolic and digestive processes gradually restore equilibrium. The same happens in fear and anger, the two "emergency" emotions mentioned by Cannon. In all these cases, emotion is a sign of disturbance and equilibrium has to be restored after the emergency is over.

Effect and reaction. Not every organismic change that follows an environmental disturbance is a homeostatic reaction. Some organismic processes are the *effect* of disturbance rather than the organismic *reaction* to it. Before a homeostatic reaction can occur, the disturbance must be registered and sensed in some way. The environment must act on the body and upset its balance before steps can be taken to restore it. Exposure to cold must affect us adversely in some way before a reaction can set in that will adapt us to lower temperatures. The adaptation is to the environment, but the homeostatic reaction is the sequel to an experienced upset. While the reaction to cold may be homeostatic and adaptive, the primary effect of cold on the body is not. During brief and partial exposure to cold (putting one hand into cold water), the lowered skin temperature of the hand and its constricted blood vessels are the effects of cold which upsets the body's temperature balance. After the hand is withdrawn, the dilatation of blood vessels in both hands is a homeostatic reaction brought about by a central mechanism which restores the balance.

Homeostasis and adaptation. Not every homeostatic reaction is adaptive. In some cases, it can be definitely harmful to the organism. Hemorrhage, for instance, lowers blood pressure; lowered blood pressure in the carotid sinus reflexly excites the sympathetic nervous system which raises the pressure again—undoubtedly a homeostatic reaction. But together with this rise in pressure, sympathetic excitation brings about greater permeability of the capillaries, probably as a result of vasoconstriction, so that fluid is lost to the surrounding tissue (see Ch. 7). This loss of fluid, added to the amount lost through bleeding, reduces the blood volume in circulation; the blood pressure falls, and the vicious circle is repeated until man or animal dies in shock. When the homeostatic reaction to hemorrhage is prevented by sympathectomy or injection of a sympathetic blocking agent, shock does not develop even though the blood pressure is lower than in normal animals (Gellhorn, 1943).

Homeostatic mechanisms seem to be adaptive only when they are adequate to counteract the disturbance. If the disturbance continues and the organismic reaction is insufficient to restore the balance, it can become harmful because of incidental secondary effects which upset the organismic balance anew. Thus homeostatic mechanisms cannot be adaptive in themselves. Adaptation results from an effort to cope with the environment in which only the outcome counts. If every organismic process were a survival reaction, if every environmental pressure inevitably resulted in an adaptive response, how could we explain the host of reactions that obviously do not result in survival? The shock reaction, the proliferation of cancer cells, coronary thrombosis or tuberculosis are neither adaptive nor survival reactions. Neither is an anxiety neurosis or a psychosis. In cases such as these, there is a continuing disturbance of normal functioning which cannot be overcome by the organism alone. It must have help from the outside if it is to survive. Unless the "wisdom of the body" is supplemented by the wisdom of another healer, the destructive process may become irreversible.

The distinction between a symptom of disturbance and the reaction to it has important consequences for our understanding of the role played by the autonomic nervous system. Either branch of this system may mediate or aggravate the *effect* of environmental stress or mobilize the *reaction* to it. In fear, for instance, the central excitation of the flight pattern includes activation of the sympathetic nervous system and adrenaline secretion. This will induce bodily changes that disturb normal equilibrium. The homeostatic reaction must counter these changes if the individual is to remain healthy. There are also disturbances that may be mediated by other systems and require a compensatory excitation of the sympathetic nervous system. This can best be demonstrated for processes in which the two branches of the autonomic system seem to be stimulated in turn, namely, during exposure to heat and cold.

HEAT AND COLD

According to Cannon, the sympathetic (adrenergic) system protects the organism from the effect of cold by constricting the blood vessels; the blood is withdrawn from the skin and shifted to the interior of the body where it is kept warm. During heat, blood vessels relax, the blood courses near the surface to be cooled and prevents the deeper regions from being overheated. This summary statement needs a good bit of qualification.

Effect of heat and cold. Surely we are not protected against cold by the withdrawal of blood from the skin. Subjectively, we feel cold as soon as the skin pales and becomes cold, whatever the cause. Cooling of the skin, fear, or adrenaline injection all produce a sensation of chilliness, sometimes combined with shivering. Similarly, we feel warm when the skin is flushed, whether that is the result of the heat of summer, a blush of embarrassment, rage, or "hot flashes" during the menopause. During a fever, the patient feels chilly when his skin is pale, and hot when it is flushed, whatever his rectal temperature.

When the air temperature rises to 100° F. during a heat wave, or when the skin is exposed to the hot sun, the blood cannot be cooled by coursing near the surface; yet that is precisely the time when there is a dilatation of the blood vessels in the skin. Actually, we seem to be protected against heat by a secondary vasoconstriction. The flush that appears during the first day of a heat wave or the first few days in the tropics soon gives way to "tropical pallor."

When we are exposed to severe cold, we are not protected against it by having the blood kept warm inside the body, but by a brisk flow of blood that will warm the exposed limbs. The increased heat production induced by shivering, or the vigorous exercise to which we are urged when we feel cold, keeps blood and tissues warm. The muscular activity accelerates blood flow and leads to a mild vasodilatation. If that is not sufficient to keep the limbs warm, there will be extreme discomfort, frostbite, and eventually death.

Experimental evidence. The primary effect of *cold* seems to be a constriction of blood vessels in the skin which extends well beyond the area cooled (Cooper and Kerslake, 1955). Such reflex vasoconstriction during cold produces a 12 percent decrease in plasma volume (D'Amato and Hegnauer, 1953), which contributes to the harmful effect of cold. In Raynaud's disease, the fingers and toes are extremely sensitive to cold and become pale, cold, and numb even on moderate cooling. After the sympathetic nerves are excluded, reflex vasoconstriction is prevented and the fingers remain pink and warm. Cannon (1932) and later workers found that animals are able to withstand exposure to cold after total sympathectomy, even though this operation prevented piloerection. Increased shivering enabled the animals to tolerate the cold without permanent ill effects. Far from counteracting the effect of cold, adrenaline has been found to aggravate it (Hartmann *et al.,* 1931).

We have suggested (Ch. 6) that the effect of cold is sensed via the peripheral fibers of the estimative system which seem to initiate a reflex vasoconstriction. This is appraised as unpleasant, to be avoided, and man or animal feels urged to seek warmth or to protect himself against cold. The felt discomfort also induces shivering and, in animals, piloerection (via the action circuit connecting with the hypothalamus and extrapyramidal pathways). Apparently, the peripheral fibers of the estimative system mediate the reflex *effect* of cold via sympathetic effectors, while the central portion of the estimative system brings about a *reaction* to cold, i.e., the induction of shivering, piloerection, and an urge to brisk activity resulting in vasodilatation.

Many research findings support this suggestion. For instance, Brown *et al.* (1954) reported that the cold-adapted Eskimos have warmer hands and forearms than nonadapted people; their blood flow in hand and forearm is also increased. Among white Americans, trained cold adaptation has the same effect. At the end of training, these people feel less discomfort in the cold, shiver less, and have warmer limbs (Carlson *et al.*, 1953). Apparently, cold adaptation results in a mild dilatation of blood vessels in skin and muscles which counteracts the effect of cold.

This dilatation of blood vessels on the surface seems to result in slightly cooler deep areas. The deep forearm temperature, for instance, is lower in Eskimos than in nonadapted whites (Brown *et al.*). Therefore, the rectal temperature is not a reliable indicator of the temperature optimum. This was shown by Andersson and associates (1956). Goats exposed to continuous heat (50° C. for 15 to 20 minutes) had only a slight rise of rectal temperature (by 0.4° to 0.7° C.), though their heavy panting showed their obvious discomfort. On the other hand, goats exposed to cold (—5° C.) had a rectal temperature 1° to 1.5° C. *higher* after one and a half hours in the cold than they did at room temperature. Yet they were extremely uncomfortable, and shivered violently during exposure and for five minutes after they had been brought back into a warm room. The rectal temperature, like deep arm or leg temperature, is very likely lower in cold-adapted people during exposure to cold than in those not cold-adapted. Of course, on continuing exposure the organismic reaction will not be sufficient to compensate for the effect of cold, and surface as well as rectal temperature will drop abruptly.

The effect of *heat* seems to be a general dilatation of blood vessels. When one leg is heated, there is a reflex vasodilatation in the hand also, which is abolished by lumbar sympathectomy; but stimulation of the lumbar chain produces vasoconstriction in the hand. This means that such reflex dilatation is mediated over the sympathetic chain and inhibits vasoconstrictor impulses (Cooper and Kerslake, 1955). Heat also results in increased blood flow and plasma volume (Hertzman, 1953). Since blood vessels respond to acetylcholine with relaxation, to adrenaline or noradrenaline with constriction, it would seem that reflex dilatation must be a cholinergic symptom. Gellhorn (1943) suggested some time ago that heat stimulates primarily the parasympathetic nervous system, while cold stimulates primarily the sympathetic nervous system. Since then it has been found that vasodilatation is carried over the sympathetic nervous system, though by cholinergic fibers (see Folkow, 1955). Sweating also is mediated by nerves in the sympathetic chain. The sweating from heat is mediated by cholinergic nerves while emotional sweating is mediated by adrenergic fibers (Shelley and Hurley, 1953). It seems that the assumed antagonism of sympathetic and parasympathetic nervous systems really amounts to reciprocal innervation of adrenergic and cholinergic fibers, whether they belong anatomically to the sympathetic or parasympathetic nervous system.

We would suggest that the *effect* of heat is mediated by fibers of the estimative system via reflex connections with cholinergic nerves, and includes vasodilatation and sweating. The *reaction* to heat would be vasoconstriction and also a general attempt to rest and cool off. This interpretation would explain why sympathectomized animals are able to withstand cold but succumb to heat. Cannon's (1932) sympathectomized monkeys died of heat prostration at a temperature that did not harm normal animals. After exclusion of the vasoconstrictors, the reaction to heat is impossible and the effect of heat continues unchecked. During cold, lack of these nerves prevents the vasoconstriction which is the effect of cold. The constant mild vasodilatation apparently favors normal flow of blood through the extremities and so reduces the effect of cold.

The autonomic nervous system in heat and cold. We have argued that the sympathetic nervous system does not always restore the homeostatic balance after a disturbance. Rather, peripheral adrenergic excitation constitutes the effect of one kind of disturbance (for instance, cold or fear) and a cholinergic reaction is needed to restore homeostasis. Other types of disturbance (for instance, heat or anger) are mediated via cholinergic nerves and need an adrenergic reaction before equilibrium can be reestablished. We shall see in the next section that these two types of stress also affect the endocrine glands in a characteristic way.

SELYE'S CONCEPT OF STRESS

Cannon's concept of homeostasis was later extended by Hans Selye in a number of papers from 1936 on. Selye noted that animals exposed to various types of stress showed not only an increased secretion of adrenaline but also an increased secretion of adrenal cortical hormones ("corticoids") which seemed to counteract the damage; hence, he suggested, there is an organized pattern of biological reactions to stress.

The reaction to stress. According to Selye, this common response to stress, superimposed upon all specific effects, "represents the somatic manifestations of non-specific 'stress' itself." Since the resistance to stress is increased during this reaction, Selye suggests that "perhaps in man also all the systemic diseases, indeed all the stresses and strains of normal life, are met by a similar defensive corticoid production. Thus developed the realization that here we are dealing with one of the fundamental adaptive reactions of vertebrate organisms" (1950, p. 3). Selye conceives of the production of adrenal cortical hormones as the fundamental defensive reaction that is set off by the stimulation of the anterior pituitary gland during stress. This gland discharges adrenocorticotrophic hormones (ACTH) into the blood stream, which stimulate the adrenal cortex to secrete various hormones. Two types of corticoid activity are important for reactions to stress. One type raises blood sugar and counteracts inflammation; hormones that show mainly this type of activity are called *glucocorticoids* (G-Cs) or *anti-inflammatory corticoids* (A-Cs). Cortisone and cortisol belong to this group. Another type brings about the retention of sodium and the excretion of potassium, and also induces or promotes inflammation; Selye calls hormones with this type of activity *mineralocorticoids* (M-Cs) or *proinflammatory corticoids* (P-Cs). Aldosterone and desoxycorticosterone belong to this group (see Selye, 1956).

Inflammation, Selye argues, is a defensive reaction of the organism because it walls off the invading organism or substance from the rest of the body. But when this substance is harmless, inflammation interferes greatly with normal functioning, which can be restored by the anti-inflammatory hormones. This happens in arthritis, for instance, where inflammation prevents movement of the joints and leads to crippling disability. However, in some cases the proinflammatory hormones, in others the anti-inflammatory hormones, can themselves produce disease. By counteracting tubercular inflammation, for instance, cortisone may allow tuberculosis to spread and devastate the whole body. Selye suggests that some diseases may result from excessive or abnormal adaptive reactions, and these diseases might be called "diseases of adaptation."

In summary, Selye contends that:

1. Any systemic stress (i.e. one which affects large parts of the body) elicits an essentially similar syndrome.

2. This syndrome helps adaptation; he calls it the "General Adaptation Syndrome" (G-A-S).
3. Adaptation can cause disease.

The types of stress Selye investigated at first were mostly of the near-fatal kind. Most of his early examples and many of his explanations apply to the most severe kind of stress, namely, traumatic shock or shock from loss of blood. Accordingly, his description of the various stages in the general adaptation syndrome is also geared to states of extreme distress.

The general adaptation syndrome. The first phase of this syndrome is the *alarm reaction* (A-R) which is defined as

> the sum of all non-specific phenomena elicited by sudden exposure to stimuli which affect large portions of the body and to which the body is quantitatively or qualitatively not adapted. Some of these phenomena are merely passive and represent signs of damage or "shock;" others are manifestations of active defense against damage. In the event of moderately severe systemic stress, from which recovery is possible, the signs of injury usually precede the appearance of defense phenomena. (Selye, 1950, p. 10)

We note in passing that Selye recognizes the difference between the passive effect of the disturbance and the active organismic reaction. Though he includes both under the "alarm reaction," he calls the passive effect the "shock phase," and the organismic reaction which follows, the "countershock phase." The shock phase is characterized by various signs of injury: reduction of temperature, lowering of blood pressure, decrease in muscular tone, loss of fluid from blood into tissues, increased permeability of capillaries and cell membranes, generalized tissue breakdown, a loss of blood chlorides with an increase of blood potassium, and a disturbed acid-base balance (acidosis). There is also a short rise (followed by a fall) in blood sugar, a decrease of white blood corpuscles followed by an increase, a decrease of eosinophil cells in the blood, and acute gastric erosions. According to Selye, the discharge of ACTH from the anterior pituitary gland, inducing the secretion of adrenal cortical hormones, is an early defense reaction; it really ushers in the second phase of the alarm reaction, the phase of countershock.

In this second phase there is an enlargement of the adrenal cortex and increased adrenal cortical secretion, a destruction of lymph cells in the thymus and lymph glands, and various other changes that reverse the effects of the first phase. Now there is a rise in blood pressure, an increase in blood chlorides and blood sugar, a rise in blood volume, a change in the acid-base balance toward the alkaline side, increased urine excretion, and often a rise in temperature.

Obviously, some of the changes described in both the shock and countershock phase are symptoms of near-fatal stress. For this kind of stress, there is a second stage after the alarm reaction, the *stage of resistance.* For Selye, the countershock phase actually is the transition to this second or resistant stage. If exposure to stress continues for some time but stress is not excessive, the countershock is stabilized at the resistance stage. This resistance stage

> is mainly characterized by an increased resistance to the particular stressor agent to which the body has been exposed and a decreased resistance to other stimuli. Thus the impression is gained that during the stage of resistance, adaptation to one agent is acquired "at the expense of" resistance to other agents. (Selye, 1950, p. 12)

Finally, when exposure to stress is continued too long or the stress is too severe, there comes the third stage of *exhaustion.* This is the result of prolonged overexposure to a particular kind of stress. Adaptation wears out and many symptoms of the alarm reaction reappear: for instance, gastrointestinal ulcers, destruction of lymph cells, and consequent shrinking of thymus and lymph glands with increased adrenocortical secretion.

This three-stage process is called the general adaptation syndrome because:

> The reaction is *general;* it is elicited only by those stresses which are generalized (in that they affect large portions of the body) and it in turn evokes generalized systemic defense phenomena.
>
> The reaction is *adaptive;* it helps the acquisition and maintenance of a state of inurement.
>
> The reaction is a *syndrome;* its individual manifestations are co-ordinated and partly even inter-dependent. (Selye, 1950, p. 12; Selye's italics)

This concept of stress and of the alarm reaction to stress has been widely applied to the field of emotion. Not only has stress been taken to mean the external cause of an emotion (so providing us with a new version of the conflict theory of emotion), it has also been taken to be the result of emotion. Stress results in emotion, but emotion can also produce stress.

The concept of stress has been used particularly for psychosomatic disturbances. It has been argued by some theorists that stress results not only in "diseases of adaptation" but also in a host of physiological changes that would be appropriate as a response to physical stress but are misplaced when stress is purely subjective. The increase of muscular tension, blood pressure, and heart rate during emotion, which is an adaptive reaction to physical stress, becomes maladaptive as a reaction to psychological stress. Hypertension as a result of emotional disturbance, or an anxiety neurosis, would be a "disease of adaptation" in Selye's sense because there is too much adaptation for a comparatively mild stress.

A whole symposium was designed to discuss the problem of "Life stress and bodily disease" (Wolff, 1950) from every conceivable angle. Several of the contributors pointed out that "stress" seems to be conceived so broadly that it includes practically every situation in which a human being may find himself. Frank A. Beach, for instance, complained:

> Performing mathematical problems is considered stressful and sweating in response to a high temperature also constitutes a stress response. . . . If the word is going to refer to everything from homeostatic mechanisms dependent upon the sympathetic nervous system to cerebral activity, which I presume is involved in mathematical calculations, we are apt to arrive at a very inclusive but equally indefinite concept of the relations between "life stress and bodily disease." (1950, p. 119)

And H. W. Smith, answering Beach, was at least half serious when he said:

> I would reply . . . that one might conceive that the only normal condition for man is in the horizontal position in a soft bed, asleep and without dreams. Everything over and above that is possibly stress. (1950, p. 120)

Unfortunately, Selye (1950) has reported that bed rest is stress, too, because it results in symptoms of the generalized alarm reaction!

Definition of stress. Obviously, the notion of stress, at first applied to extreme disturbances, has come to be used for conditions encountered by any living organism in its everyday existence. Such usage, as Beach pointed out, is too wide to be useful. We propose to examine this concept and define it before we try to discover its relation to emotion.

Selye first defined stress as "the biologic equivalent of physical stress"; it is the result of "the interaction between a force and the resistance opposed to it." But what is the "biologic equivalent" of physical stress? In the functioning of any organism and even in the functioning of a machine, there is always an interaction between a force and the resistance to it. Breathing, opening or closing the eyes, speaking, swallowing, even standing, all involve an interaction between force and resistance. Organismic processes like blood flow and digestion also involve mechanical force and resistance.

Later, Selye tried to define the concept of stress more exactly:

> Stress is the state manifested by a specific syndrome which consists of all the nonspecifically induced changes within a biologic system. (1956, p. 54)
>
> Stress is the sum of all wear and tear caused by any kind of vital reaction throughout the body at any one time. That is why it can act as a common denominator of all the biologic changes which go on in the body. (1956, p. 274)
>
> The condition of biologic stress is essentially an adjustment, through the development of an antagonism between an aggressor and the resistance offered to it by the body. (1956, p. 55)

In the first definition, Selye uses an operational approach, defining stress as *the state* that shows a stress syndrome; in the second, he seems to say that it is *the vital reaction* to environmental pressure that causes the wear and tear we call stress. And in the third, he seems to imply that stress is *the outcome* of various vital reactions, the completed adjustment. Since these definitions do not seem to be consistent, let us see whether we can arrive at an understanding of "stress" that might resolve the difficulties.

The term "stress," like so many terms in physics, was originally taken from subjective experience, that of either suffering or exerting *special* force (as "putting stress on a word," "suffering distress"). Stress did not apply to the normal resistance of everyday functioning but to special conditions that must be met by special exertion. In physics, the terms "stress" and "strain," which in subjective experience are practically interchangeable, were given special meanings. There is no reason why physics or any other science should not use common terms with specialized meanings adapted to their own subject matter. But when we reintroduce these terms into psychology or biology with the special meaning the physicist has given them, the result will be confusion.

The biological equivalent of the physicist's definition of stress cannot be the force and resistance involved in normal functioning, or we would have to coin a different term for the stress that disturbs normal functioning and brings about a stress syndrome. Normal functioning must include smooth functioning on the psychological as well as on the physiological level. On the physiological level, smooth functioning requires not only the integrated activity of various organ systems but also a normal rhythm of waking and sleeping. During waking, the minimal activity of the organism as a whole is to find food and eat it and thus maintain life; thus normal functioning must include goal-directed action. Such action means that something is *wanted* and so implies emotion. In the human being, goal-directed action also implies planning and thinking. These psychological activities, too, must be included in that interlocking chain of balanced activity which is normal functioning.

A normal round of such balanced activity will be maintained without stress if activities on every level of functioning go on smoothly, and are properly articulated and interrelated. When planning meets no obstacle, there is no stress, no sense of pressure, and no necessity for extra effort. If a goal has to be reached but plan after plan miscarries, we are aware of difficulty, of stress and frustration. We may react with increased effort, or with emotion, or both. On the physiological level, stress is experienced as upset or disturbance. Every organ and organ system functions smoothly when it is healthy and is supplied with the proper materials for its activity. But if a boy eats green apples, there is undue stress on his digestive system; it is disturbed, upset, and most unpleasant subjective experiences will follow. On the biochemical level, normal functioning means a dynamic balance between various hormones and enzymes. Let one of these secretions drop out or be present to excess, or give an injection that upsets the balance, and immediately there will be symptoms of disturbance, of stress. These symptoms will produce unpleasant subjective experiences as well. On every level, stress includes a subjective experience of discomfort and unpleasantness, while normal functioning in the healthy person is accompanied by a feeling of well-being.

If this analysis is correct, stress could be called any condition that disturbs normal functioning. The organismic reaction to stress is that *extra*-ordinary, *intensified* activity that is required to counteract the disturbance and restore normal functioning. Such extraordinary activity often includes goal-directed action: pain arouses a desire to escape which is carried out if possible; stimulant drugs arouse general activity, depressants induce general relaxation; and this, in addition to the organic changes that are usually meant when we speak of the "stress syndrome." Such a definition of stress as a disturbance of normal functioning, an extra load to be handled, is in agreement with common usage and also with Selye's original use of the term. He first discussed stress situations that certainly tax the body's resources. During shock, the organism is all but overwhelmed and will rally only if it can marshall vital reactions that will eventually produce resistance.

ORGANISMIC DISTURBANCE AND COUNTERACTION

For the sake of clarity it is useful to distinguish between the changes occurring as a passive *effect* of the disturbance and the changes occurring as a result of the body's *extraordinary activity* to counteract the damage. Selye, as mentioned before, included both the damage and the repair under his "alarm reaction." He emphasized that every stressor may have its particular "specific" effects, but that stress must also affect the organism as a whole, else there could be no "general alarm reaction." If that is so, there must be some way in which a given stressor affects the body. Selye suggested that either the nervous system or the circulation or both must carry impulses from an injured part to the rest of the body, to give the alarm and arouse the vital reaction.

We have suggested before (Ch. 6) that the peripheral and spinal fibers of the estimative system mediate the *effect* of some types of stimulation by vasomotor reflexes. Adrenergic stimulation seems to represent the effect of *cold* and includes vasoconstriction in the skin together with adrenomedullary secretion which releases glycogen from liver and muscles and so increases the blood sugar. The effect of *heat,* on the other hand, seems to be mediated via cholinergic vasodilatation. The reaction to both cold and heat seems to be initiated from the central portions of the estimative system via the hypothalamic branch of the action circuit.

But there must also be a way in which the blood can be directly affected by stress, and a way in which this effect can be registered and counteracted. The composition of the blood can be affected by diet, for instance. Eating salty food increases the sodium chloride concentration in the blood; drinking a great deal of water decreases it. We have argued (Ch. 6) that the sodium content of the blood can be detected in the hypothalamus (perhaps via the pituitary portal system), and that increased sodium will activate a neural relay that will initiate a felt impulse to drink (thirst) and also the secretion of antidiuretic hormone to prevent water loss. Decreased sodium content will activate neural relays to the olfactory estimative areas to intensify the appeal of salt and arouse an impulse to eat it. The content of adrenal cortical hormones in the blood seems to be detected in a similar way and to result in the increased or decreased secretion of ACTH, to provide sufficient glucocorticoids for carbohydrate metabolism. This would account for the fact that the rate of ACTH secretion ordinarily depends on the amount of glucocorticoids available.

According to Selye (1956), the composition of the blood is changed by injury and this change can induce increased secretion of ACTH. When the hind limb of a deeply anesthetized rat is completely separated from the body (except for the blood vessels), mechanical injury or scalding of that limb produces the "alarm reaction" with adrenocortical stimulation. On the other hand, Anderson and coworkers (1957) have reported that dogs with complete midbrain transection do not show adrenocortical activation even when they are severely injured (by scalding or laparotomy). We doubt that species differences can account for this discrepancy. It is more likely that local vasomotor reflexes are induced in the severed limb via afferents in the arterial walls which connect with the sympathetic chain.

This would mean that the effect of many types of stress is mediated via connections from peripheral nerves of the estimative system to the autonomic system; and that the effect of stress is then sensed and appraised via sensory and estimative fibers from the skin, blood vessels, and other structures that are affected by these autonomic changes. We like or dislike the *effect* of warmth or cold, but we are not aware of the way in which it is produced, for subthalamic activity of the estimative system is reflex in nature. Perhaps the effect of drugs and hormones is an exception. We have suggested (Ch. 5) that various drugs affect specific pathways in the brain and eventually either stimulate or depress the action circuit and so may stimulate or depress pituitary secretion. Thus adrenaline stimulates ACTH secretion while reserpine and chlorpromazine depress it (Kayser, 1957).

Accordingly, we assume that many types of stress affect the body via local reflexes and are counteracted via central pathways. But do the various types of stress affect the body always in the same general way, as Selye says? Or do the two pathways in the hypothalamus, one serving the reaction to cold, another the reaction to heat, imply a different pattern of hormone secretions? Though the reaction to stress includes a host of biochemical changes, we shall confine ourselves to tracing the changes in endocrine secretion during stress, the changes that led Selye to his concept of a general adaptation syndrome.

Endocrine changes during cold. Exposure to cold results in increased sympathetic excitation and adrenaline secretion (see above; also Malméjac *et al.*, 1956). This produces a decrease in liver and muscle glycogen, provided glucocorticoids are available (Hechter, 1955). These, we believe, are the *effects* of cold.

The organismic reaction, mediated via the central portion of the estimative system, seems to consist of the release of ACTH from the anterior pituitary gland which induces the discharge of adrenal cortical hormones with glucocorticoid activity. Both adrenaline and glucocorticoids inhibit glucose utilization by bodily tissues (Bacila and Barron, 1954; Bullough, 1955). As a result, cell division is inhibited in lymph glands and spleen; and this shrinking of spleen and lymph glands is characteristic for Selye's first stage of the general adaptation syndrome. Glucocorticoids inhibit the glycogen depletion in the muscles that is caused by adrenaline (Wortman and Leonard, 1953) and so prevent muscular weakness; they also promote gluconeogenesis from protein. Both these effects account for the fact that glucocorticoids are necessary for maintaining muscular strength.

The discharge of ACTH also results in a decrease of serum chlorides and sodium and an increase in blood potassium and phosphates (Selye, 1950). This seems to be compensated by an increased secretion of mineralocorticoids which induce sodium retention and potassium excretion. Thus both glucocorticoids and mineralocorticoids seem to be necessary for the organismic reaction to cold. This is shown, for instance, by Hartmann *et al.'s* report (1931) that rats were unable to withstand cold after removal of their adrenals. When they were maintained on a high salt diet (thus replacing serum chlorides and sodium), their resistance to cold increased to some extent. When they were supplied with cortisone (which has mainly glucocorticoid activity), they reacted like intact animals. But when salt-maintained rats were given adrenaline before they were exposed to cold, all but one died either during the test or a few hours afterward. Apparently, adrenaline aggravates the effect of cold because it intensifies sympathetic excitation.

The rise of blood sugar during cold (the result of the mobilization of glycogen from liver and muscles) is counteracted by the discharge of somatotrophic hormone (STH) into the blood stream, which induces the secretion of insulin from the islands of Langerhans in the pancreas (Bullough, 1955). This promotes utilization of the blood sugar and results in normal carbohydrate metabolism. When glucose is given during cold, or food intake is increased, cold is better tolerated (Hertzmann, 1955). So long as sufficient glucose and insulin are available to counteract the effect of cold, and so long as the central relays that mediate the organismic reaction are functioning, normal equilibrium will be maintained. The normal cell division in lymph glands and spleen is restored and these organs will return to their normal size. This is the resistance stage of Selye's G-A-S.

But if the cold is extreme or continues for long periods, the organismic reaction (which includes thyroid-stimulating hormone, TSH, to increase metabolism) is not sufficient to counteract the sympathetic effects. Eventually, extreme cold seems to depress and finally to paralyze the multineuronal chains of the estimative system, for responses to sensory stimulation are increased while pain is depressed (Kayser, 1957). This means a gradual depression of the organismic reaction. This stage is again characterized by an inability to utilize glucose, so resulting in the shrinkage of spleen and lymph glands Selye has noted in the third, exhaustion stage of the G-A-S.

Endocrine effects of fear. In fear, the appraisal of harm to come arouses an impulse to escape, which is accompanied by a stimulation of the anterior pituitary gland and followed by the secretion of adrenocortical hormones. Since fear also induces the stimulation of the sympathetic nervous system, its effects on the body will resemble the effect of moderate exposure to cold. The secretion of adrenocortical hormones takes one to two hours to come to its peak (Thorn and associates, 1953), which means that this reaction is too late to facilitate immediate flight. Rather, it is needed to reestablish homeostasis after the danger is past. When fear becomes chronic, there is no longer the same powerful impulse to escape; instead, there is a cumlative effect of sympathetic stimulation, the ACTH discharge becomes minimal, and the effect of fear is now unopposed. This results in the reduced muscular efficiency and incapacity for sustained work found by Cohen and White (cf. Ch. 7).

Endocrine changes during heat. During heat, a vasodilatation in skin and other tissue, an increase of the heart rate, and increased blood flow is produced reflexly (Cooper and Kerslake, 1955). Sweating also seems to be produced by a local reflex. All these effects of heat, we think, are produced via peripheral and spinal fibers of the estimative system connecting with cholinergic fibers of the sympathetic system.

The general vasodilatation reduces the blood pressure and requires an increased blood volume. Apparently, stretch receptors in the right atrium of the heart can register the reduced fluid pressure (see Henry and Pearce,

1956) and so induce the discharge of a hormone (via connections to neurosecretory cells in the diencephalon), which stimulates the secretion of aldosterone from the adrenal cortex. Aldosterone is a mineralocorticoid and prevents the excretion of sodium (Davis *et al.*, 1956), thus raising the sodium content of the blood. This rise seems to be detected by receptors in the hypothalamus which induce thirst and also the discharge of antidiuretic hormone (ADH) from the posterior pituitary gland. Now the water taken in is retained and makes good the fluid lost in sweat and the extra fluid required by the vasodilatation. So long as the heat continues, and the blood volume needs to be replenished, aldosterone and ADH secretion will go on, making it possible for the organism to sustain the effects of heat.

The hormone that stimulates aldosterone secretion seems to be the somatotrophic hormone, STH; apparently, its secretion is independent of the anterior pituitary gland, for it continues to act even though anterior pituitary function (and ACTH secretion) is lost. If cortisone is supplied after destruction of this gland, together with sex hormone and thyroxine, the normal state is reestablished; body hair regrows, the skin loses its fine wrinkled thinness, and there is a decided weight gain (Astwood, 1955). Somatotrophic hormone prevents sodium loss (Carballeira *et al.*, 1952) and is discharged into the hypothalamic portal veins even after the anterior pituitary gland is removed (Long, 1956). These facts seem to indicate that STH stimulates growth (by stimulating insulin secretion, according to Bullough, 1955) and also induces the secretion of sodium-retaining aldosterone. Its increasing secretion during heat would facilitate the utilization of glucose; this would account for the reduced need for food, and also for the increased size of lymph glands and spleen (through increased cell division), which has been observed in animals exposed to intense heat (Selye, 1950). This effect on spleen and lymph glands can be duplicated by the administration of adrenal cortical hormones with mineralocorticoid activity.

The organismic reaction to the effect of heat and to the associated changes seems to be a secondary vasoconstriction and the associated discharge of glucocorticoids. Actually, glucocorticoids seem to be beneficial in heat stroke and artificial fevers (Selye, 1950). This does not mean that mineralocorticoids are not necessary during the exposure to heat. But to supply them from outside would aggravate the effects of heat by increasing the sodium retention and inducing more and more thirst. Normally, the secretion of STH will be arrested as soon as the increased blood volume activates the stretch receptors in the atrium.

Endocrine effects of anger. Anger seems to produce an effect similar to that of heat. The flush of hot anger is not restricted to the face. The gastric mucosa reddens, swells, and becomes congested to the point of causing actual hemorrhages (Wolff, 1950). Thus anger and rage, like heat, seem to imply cholinergic excitation. These emotions are also accompanied by noradrenaline secretion. Van der Pol (1956) reported that noradrenaline does not induce the discharge of ACTH, which would mean that anger does not have the same effect as fear, nor does it arouse the same reaction. But in anger, as in heat, STH secretion will continue as long as greater blood volume is needed, and will have the usual effect on cell division and sodium retention.

Emotion and stress. When emotion is identified with stress, it is assumed that every emotion must be a disturbance. But some emotions, like joy or happiness, may actually improve human functions and bring them closer to the optimum.* There are others, particularly fear and anger, that do interfere with normal functioning. Nothing is gained, however, by calling all emotions "stress." Rather, we must discover which emotions disturb the organism and in what way they interfere with normal functioning. We have attempted a summary of the physiological effects of emotion in this and the preceding chapter, though there are experimental data only for fear and anger. We hope to discuss the psychological effect of disturbing emotions in Chapter 9.

The more intense the emotional disturbance, the more widespread will be its effects and the more general will be the organismic reaction. Some of the effects will continue even after the organismic reaction has begun. As a result, the literature is filled with contradictory reports: that a given stress produces high or low blood sugar, sodium excretion or sodium retention, etc. Only when the exact sequence of changes is recorded, can we decide whether a given phenomenon is the effect of stress or the reaction to it.

It is no accident that there are similarities between anger and heat, or between fear and cold. Every primary cholinergic excitation (e.g., mecholyl injections, heat, anger, love) seems to arouse a sensation of warmth;

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* For a discussion of normal functioning as optimal functioning, see Arnold and Gasson (1954), p. 390.

every primary adrenergic excitation (adrenaline injection, cold, fear) seems to induce a feeling of cold. Even the secondary adrenergic reaction to a primary cholinergic excitation may induce a sensation of cold (chills after mecholyl injection as soon as the blood pressure rises again), just as the secondary reaction to a primary adrenergic excitation may bring with it a sensation of warmth (after the first chill has passed on taking a cold plunge, there is a feeling of comfortable warmth). When either primary cholinergic or primary adrenergic stimulation is severe, there may even be a reaction to the reaction before the normal balance is reestablished: coming into a warm room out of the cold, we first feel excessively warm, then we may begin to feel chilly again until finally a comfortable warmth prevails. There seem to be similar fluctuations in endocrine secretion, for investigators often report biphasic or triphasic physiological changes during stress.

Chronic stress. When stress is intense, the return to normal equilibrium will take much longer. During the compensatory reaction, the primary effect will gradually be counteracted until normal balance is achieved, though that may be a matter of days or weeks (for instance, in cold adaptation). When the compensatory reaction is fully developed and stress is not too intense, the organism approaches normality. When stress is so intense or so prolonged that the vital reaction cannot restore normal functioning, the primary effects will reappear again in full force and the damage will increase until it ends in death.

According to Selye, every type of stress produces essentially the same three stages of organismic reaction in the general adaptation syndrome. He ascribed the differences often found in the initial phase to specific effects of the stressor or to such "conditioning factors" as heredity, previous exposure to various stressors, diet, and the like.

It can easily be shown, however, that the general effects of stress show certain similarities among a whole group of stressors. Extreme fear, exhausting exercise, cold, and adrenaline have similar primary effects because they are all adrenergic stressors, affecting primarily the sympathetic nervous system. Likewise heat, anger, mecholyl, and insulin have similar primary effects because they are all cholinergic stressors with effects opposite to those of the adrenergic stressors.

Since the primary effect of adrenergic stressors differs from that of cholinergic stressors, the final phase of exhaustion should be different also. This is shown by the opposite effects of various types of stress on the thymus gland, lymph nodes, and spleen, the organs most dramatically affected by stress of all kinds. During exposure to *cold* or forced exercise, for instance, a marked *shrinking* of these organs is found after forty-eight hours. During the resistance stage, their size increases again to approximately normal; but in the exhaustion stage thymus and spleen shrink even more than they did during the first stage. During exposure to *heat,* on suffering burns or fever, the spleen is markedly *enlarged,* becomes normal on recovery, but shows a further enlargement if the animal succumbs (Selye, 1950, pp. 460, 462). Thus the effect of acute exposure is repeated and intensified in the exhaustion phase; but there is a sharp difference during these two stages in the appearance of thymus, lymph nodes, and spleen in animals exposed to cold as compared to animals exposed to heat.

The enlargement of thymus, lymph nodes, and spleen during heat can be duplicated by the administration of mineralocorticoids, while their decrease in size during cold can be duplicated by supplying glucocorticoids. When mineralocorticoids are given for some time to a normal animal, the acute enlargement produced at first is soon followed by a decrease in size to approximately normal in the resistance stage. When treatment is continued, thymus and spleen begin to enlarge again until they are much larger than the glands of untreated animals (Selye, 1950, p. 476). The administration of glucocorticoids, on the other hand, produces involution of thymus and lymph nodes and shrinking of the spleen in the first stage; if the dose was moderate, there is a second (resistance) stage, in which these organs return to their normal size. In the exhaustion stage, finally, they become even smaller than they were in the first stage.

It would seem, then, that the discharge of glucocorticoids is induced by the reaction to an adrenergic effect of stress, while the discharge of mineralocorticoids is induced by the reaction to a cholinergic effect of stress. During any kind of stress, there is always a psychological *experience* of the effects of stress (unless anesthetics prevent it): a feeling of heat or cold, of fear or anger, of different kinds of malaise, all mediated by the estimative system. There is also an urge to escape or to deal in some way with the distressing situation. This impulse to action is mediated, we believe, over the action circuit which sends relays to the hypothalamus and the pituitary gland. Whenever a particular action pattern includes an adrenergic reaction to a cholinergic effect of stress, the anterior pituitary is apparently induced to discharge compensatory ACTH, stimulating the adrenal cortex to secrete glucocorticoids. Whenever the action pattern includes a cholinergic reaction to an adrenergic effect of stress, relays to neurosecretory cells seem to induce the discharge of STH stimulating mineralocorticoid and insulin secretion.

It seems certain that hormones that have either mainly glucocorticoid activity or mainly mineralocorticoid activity are secreted from the adrenals (Simpson *et al.*, 1952; Selye, 1956). The reason why pure activity of one type or another can never be observed may lie in the inevitable reaction that occurs whenever a particular hormone is given. Cortisone, for instance, which has primarily glucocorticoid activity, may induce a secondary reaction to compensate for its primary effect, and this reaction will have mineralocorticoid symptoms. To discover whether this is so, the time relation of the various physiological changes would have to be reported and compared. The early changes will represent the effect of stress, the later changes the vital reaction.

If our hypothesis is correct, we would expect severe adrenergic stress coupled with glucocorticoid secretion to lead eventually to a deficiency in mineralocorticoids; accordingly, it might be beneficial to supply them. On the other hand, severe cholinergic stress coupled with mineralocorticoid secretion should lead to an eventual deficiency of glucocorticoids which might be supplied from outside. Tentatively we might suggest that fear, cold, hemorrhage, asphyxia, adrenaline, thyroxine, and exhausting exercise primarily affect the sympathetic-glucocorticoid system, while heat, anger, mecholyl, histamine, insulin, morphine and other opiates, anoxia, acute inflammation, and anaphylactic reactions primarily induce cholinergic-mineralocorticoid activity. There is some evidence that mineralocorticoids are of benefit in the first group, glucocorticoids in the second group.

For instance, mineralocorticoids reduce the mortality from exhaustion after strenuous exercise in normal animals and from fear of electroshock in adrenalectomized animals; wild strains of rats (more fearful, hence presumably more apt to have an alarm reaction) need a much larger dose than laboratory rats. The toxic effects of thyroxine are counteracted by mineralocorticoids, both in normal and in adrenalectomized guinea pigs and mice. On the other hand, glucocorticoids improve the work performance of rats exposed to heat, but have no effect on rats working at room temperature. Glucocorticoids have been found helpful in burn shock and heat stroke but are of doubtful value in shock from exposure to cold (see Selye, 1950).

Adrenaline and, even more, glucocorticoids are beneficial in anoxia, as well as in insulin, histamine, and anaphylactic shock; glucocorticoids are beneficial in peptone shock while mineralocorticoids are not (Selye, 1950). That the effect of anaphylactic shock is at least partly mediated via the parasympathetic system is shown by Mikulicich and Dester (1952), who found that bilateral vagotomy in rabbits decreased their sensitivity to anaphylaxis to such an extent that none of them suffered general shock; the acute heart changes usually seen in the electrocardiogram were depressed and sometimes abolished. On the other hand, when the spinal cord was cut and the sympathetic reaction thus prevented, the sensitivity of the animals was increased; now every animal suffered anaphylactic shock. Sympathectomy also decreases the resistance of dogs to histamine, acetylcholine, and peptone and seriously lowers the resistance of cats to anoxia (Selye, 1950).

Resistance and sensitization. Once resistance to a given stress has developed, it may affect the reaction of man or animal to other types of stress. Selye mentioned that either "crossed sensitization" or "crossed resistance" may develop. If an animal has developed resistance to one stressor, but has become more sensitive to another, it shows "crossed sensitization." If the animal is less sensitive to a new stressor after it has become resistant to an earlier one, it shows "crossed resistance." Various "conditioning factors" bring about either crossed sensitization or crossed resistance. But beyond mentioning such factors (heredity, diet, emotional state, etc.) Selye provided no clue that might help to predict whether an animal will be more sensitive or more resistant to a stressor as yet untried.

If we develop our own hypothesis we find that such prediction does become possible. Resistance may be considered either as a reduced effect of the stressor or as an increased capactiy of the organism to counteract it. Now we know that some stressors affect the body via the central nervous system, as, for instance, fear, anger, and also various drugs. Sedatives reduce or prevent anterior pituitary secretion after stress. Pentobarbital, for instance, inhibits ACTH secretion after sensory stimulation, exposure to cold, and morphine injection. Morphine (after adaptation) prevents ACTH secretion after unilateral adrenalectomy, abdominal operation, or injection of adrenaline, amphetamine, histamine, or vasopressin (Ohler and Sevy, 1956). Since morphine does not block the pituitary gland directly, it seems to depress a hypothalamic pathway that stimulates the gland, either by blocking the action circuit that connects with this pathway, or by depressing the estimative system. Morphine not only dulls pain, it also produces a pleasurable languor—which would suggest that it affects the estimative system. Barbiturates depress cortical and thalamic function (see Ch. 5, Drug Effects) and thus seem to depress the action circuit as well. Stressors that stimulate the anterior pituitary gland via the hypothalamic pathway seem to produce their effect via the action circuit which is touched off by unpleasant subjective sensations mediated by the estimative system.

During adaptation, the effect of stress is reduced, not directly as on sedation, but indirectly, by raising the reactive capacity of the organism. When there is repeated stress, for instance, a repeated exposure to cold, the vital reaction to it (cholinergic, glucocorticoid, and mineralocorticoid activity) seems to improve the organismic state. The adrenal cortex enlarges and the vital reaction to cold will now be more effective. The circulation improves, resulting eventually in the permanently warmer limbs of cold-adapted people. Thus the effectiveness of the vital reaction increases with an increased capacity of the organism, acquired by repeated exposure to moderate stress. The subjective experience of stress will eventually be felt as less unpleasant simply because the improved circulation has reduced the effect of cold, and neither shivering nor the secretion of ACTH will be as pronounced as before. This gradually acquired resistance results in a reduced alarm reaction to stress. Indeed, Pincus (1945) mentions that the men most efficient under stress, the "iron men," had minimal adrenocortical secretion.

However, there is a profound difference between the resistance developed after repeated exposure to moderate stress and the resistance during intense chronic stress. When resistance develops after moderate stress, the organismic reaction has resulted in increased production and storage of adrenal cortical hormones. Any similar stress can now be combatted more effectively. When resistance develops during chronic stress, the adrenocortical system is already engaged maximally to maintain near-normal function. Any other stress demanding the same type of organismic reaction will be harmful because the reactive capacity of the organism will be overtaxed. This distinction between the resistance shown after acute stress and the resistance during chronic stress will enable us to explain some reported instances of "crossed resistance" and "crossed sensitization."

Crossed sensitization. According to Selye, crossed sensitization develops when resistance to one stressor is accompanied by increased sensitivity to another. We would suggest that the resistance acquired during repeated exposures to moderate adrenergic stress will result in increased sensitivity to cholinergic stress, and vice versa. But the resistance during *chronic* adrenergic stress will result not only in increased sensitivity to cholinergic stress but also in sensitivity to another adrenergic stress; the same applies *mutatis mutandis* to the acquired resistance to cholinergic stress.

This hypothesis finds considerable support from experimental findings. For instance, strenuous muscular exercise reduces the resistance to cold and atropine (Selye, 1950). We have seen early in this chapter that both cold and exhausting muscular exercise stimulate the sympathetic nervous system while atropine prevents a cholinergic reaction. Thus cold and atropine represent adrenergic stress; if the adrenergic stress of exhausting exercise is added, the vital reaction will fail.

It is also reported that pretreatment with morphine reduces the resistance to cold and to atropine. Morphine addicts who have acquired great resistance to the drug react more intensely than normal people to the cold pressor test (Selye, 1950). We have seen (p. 252) that morphine (after adaptation) prevents the organismic reaction to adrenaline by preventing ACTH secretion. At the same time, morphine stimulates cholinergic activity (Himmelsbach, 1944). Addiction results in habituation to cholinergic effects. As soon as the morphine effect wears off, an intense adrenergic reaction is felt which produces the trembling, pallor, and feeling of weakness that compel the addict to take another dose. Without the secretion of adrenocortical hormones induced by ACTH, this adrenergic reaction will be excessive and harmful. This accounts for the increased discomfort of addicts in the cold pressor test and their decreased resistance to atropine.

Crossed resistance. When one stressor protects against the effects of another stressor, crossed resistance appears. The alarm reaction produced by an injection of formaldehyde was found to protect animals against anaphylactic shock, provided they were given eighteen to twenty-four hours rest after the injection before inducing anaphylaxis (Selye, 1950). Formaldehyde produces irritation and inflammation, and thus seems to affect primarily cholinergic-mineralocorticoid activity. Provided there is an effective vital reaction (insured by the rest period), it will protect against another similar stressor; consequently, it protects against anaphylactic shock, another cholinergic stressor.

It has also been reported that the appendicitis-like changes produced by intravenous injection of histamine can be prevented by pretreatment with histamine. They can also be prevented by a simultaneous injection of glucocorticoids, while mineralocorticoids aggravate the histamine effect (Selye, 1950). This shows that histamine seems to produce the anaphylactic effect by mobilizing mineralocorticoids, and also, that the action of glucocorticoids represents the organismic reaction. After the first histamine injection, the vital reaction seems to consist in the increased production of glucocorticoids which are stored in the adrenal cortex and will be available for counteracting another histamine injection. Obviously, the timing of the second injection is important; if it is long enough delayed until there is a considerable store of glucocorticoids, there will be no inflammation; if the second injection comes too soon after the first one, at a time when the available glucocorticoids have been spent in defense and not yet replaced, the inflammation will be more severe than the first time; if the second injection is too long delayed, the glucocorticoid store may have leveled off again and the histamine inflammation may occur as usual.

It seems that crossed sensitization occurs when the new stressor demands an organismic reaction which is not available in sufficient quantity. Crossed resistance seems to occur when the new stressor encounters an intensified organismic reaction, built up by successful repeated responses to a similar stressor.

Diseases of adaptation. With the realization that adrenocortical secretion plays a major role in resistance to stress, came the attempt to supply such hormones from the outside. ACTH and cortisone have been used to combat inflammatory diseases (with conspicuous success in arthritis), but continuous dosage results in various symptoms of endocrine imbalance. A transient diabetes may be produced which apparently is the result of an inhibition of insulin secretion and thus of reduced glucose utilization (Franckson *et al.*, 1953). According to Selye, such imbalance appears also in the reaction to long-continued stress. The organismic defense itself may become harmful and give rise to "diseases of adaptation."

This concept has met with considerable opposition. Ingle (1952), for instance, has pointed out that overdosage of adrenocortical hormones has not produced "adaptation diseases" in normal animals. To produce such diseases, the animals must be deprived of one kidney and given an abnormally high sodium load. In other words, the endocrine mechanism must be severely impaired before stress can produce disease.

Normally, no one secretion seems to act in isolation. There is always a system of checks and balances that prevents excessive endocrine secretion or, at least, counteracts it. When there is a gross derangement of endocrine secretion, it does not seem to be the result of the general adaptation syndrome, but rather the failure of an essential reaction to it. Perhaps it would be preferable to look upon the symptoms thus produced not as an excessive reaction to stress but as the sign of a new imbalance.

The diabetes produced in animals by ACTH overdosage, for instance, seems to result from the reduced insulin secretion (which is inhibited by cortisone). But if insulin secretion is stimulated by STH, as seems likely according to some experimental results (Bullough, 1955), this would amount to a failure of the organismic reaction to ACTH, a failure to mobilize sufficient STH for the secretion of insulin, thus counteracting ACTH effects. We do not conceive of such counteraction as canceling the effects of ACTH but rather as complementing them. Both glucocorticoids and mineralocorticoids, both insulin and glucagon, are necessary for the bodily economy.

THE RATIONALE OF THERAPY

Our discussion of stress seems to show that any interference with normal functioning has a primary effect and a secondary reaction that will result in a new organismic balance. This may break down if the stress is too severe. We would prefer to distinguish between the effect of stress and the vital reaction to it as two stages concealed in Selye's "alarm reaction," with his stage of resistance as the newly achieved balance. Selye's third stage, exhaustion, only occurs with the severest types of stress and really represents the breakdown of the vital reaction.

Given the kind of disturbance that results in disease, any of these stages can be influenced therapeutically. The *effect* of the disturbance may be reduced or eliminated (by suppressing the symptoms or eliminating the cause of the disturbance); or the organismic *reaction* may be supported by supplying either glucocorticoids or mineralocorticoids from the outside (or inducing their increased secretion by giving ACTH or STH); finally, therapy may aim at achieving a new organismic *balance*. The first two types of therapy we will call *direct therapy*, because there is a direct attempt to interfere with the course of stress. The third type of therapy we will call *indirect therapy* because remedies are not used for the sake of an immediate effect but rather for the sake of the new balance that will be established when the organism reacts to them.

Direct therapy. Throughout history, medical practice seems to have preferred direct therapy; and of the two types of direct therapy, by far the older and more commonly used type is the direct suppression of the disturbance, by suppressing either its effects or its cause.

Whether aspirin is used to suppress a headache or barbiturates to reduce activity and promote sleep, whether arsenicals and fever therapy are used to kill the syphilitic spirochetes, or "wonder drugs" to combat pneumonia, the underlying aim is always to attack the stressor and eliminate the disturbance. In the same way, sympathectomy is used to prevent the sympathetic effect of cold (e.g., in Raynaud's disease), and vagotomy to abolish the effect of parasympathetic excitation (e.g., in gastric ulcer). Among psychological disturbances, anxiety is reduced by prefrontal lobotomy, psychomotor epilepsy eliminated by anterior temporal lobectomy, and obsessive-compulsive trends are reduced by cingulotomy or cingulectomy. In every case, structures which seem to mediate the effect of a particular disturbance are destroyed or isolated. Such a direct attack on the effect of stress may be undertaken by chemotherapy as well as by surgery. Anxiety can be reduced by taking barbiturates and, more recently, tranquilizing drugs. In most of these cases, there is temporary relief. Surprisingly often, there is a relapse or a period of ailing convalescence.

A direct attack on the stressor has its drawbacks. Antibiotics may prevent the proliferation of invading microbes but also the growth and nourishment of healthy cells. Barbiturates and tranquilizers depress the action circuit but also depress the organismic reaction. When their effects wear off, the vital reaction will intensify the effects of the original disturbance. A surgical interference may eliminate the source of a disturbance or some of its effects at the price of an interference with normal functioning, the more seriously, the more important the excised structure.

Therapy by direct attack on the stressor seems to be based on the notion that the organism is a passive reactive system that has been disturbed by an invasion from outside. The illness is taken as proof that the body has no effective defense. Must not the much-flouted "wisdom of the body" have turned into foolishness to allow the ravages of disease? It almost seems as if remedies were tried on the assumption that the invaders from outside, whether germs, viruses, or bacteria, can be got rid of as simply as sand can be flushed out of the gears of a machine, leaving it clean and ready for normal operation. Surely there is enough evidence to show that a living being is not a passive system. A living organism does not merely suffer the effects of external manipulation as does a stick or a stone, or even a machine; it reacts to every disturbance, to the remedy as much as to the disease.

The vital reaction to the remedy seems to explain the well-known resistance to drugs that may develop with continuous dosage. The dose must be increased to achieve the same effect, which intensifies the undesirable "side" effects. The microorganisms that multiply unchecked in disease also react to the remedy, and develop a resistance to the very drugs that are meant to destroy them. Often the invading microbes seem better able to withstand the harmful effects of the drug than does the body. Even if the remedy succeeds in destroying the invaders, the body's resistance to later infections is often dangerously reduced.

Reinforcement of the vital reaction. It might seem that a reinforcement of the reaction to a disturbance would be preferable to reducing the effect of stress. But to reinforce the vital reaction we must know what is effect and what reaction. Ever since it was discovered that adrenocortical hormones are secreted during stress, these hormones have been used to counteract the effects of various disturbances. Both ACTH (which induces the secretion of glucocorticoids) and cortisone (with glucocorticoid activity) have been successful in alleviating diseases like rheumatoid arthritis, but have been disappointing in many others. However, such therapy is not without danger. At first, the increased supply of these hormones produces a decided feeling of well-being, of buoyance, but this may eventually turn into a severe depression. We venture to suggest that the feeling of well-being lasts as long as the increased supply of glucocorticoids is balanced by an organismic reaction that induces STH discharge and the secretion of insulin. With long-continued therapy, this reaction subsides. Continuous use of either ACTH or cortisone depresses thyroid function and arrests growth, and lymph glands and spleen shrink—all signs that the vital reaction is insufficient to restore the normal balance. The well-known susceptibility of cortisone-treated patients to virulent infections is a further indication that they live in a dangerous state of imbalance.

The use of insulin therapy in anxiety states is another example. The additional supply of insulin seems to counteract some of the effects of fear. Whether given in shock or subshock doses, insulin increases glucose utilization and stimulates carbohydrate metabolism in the brain as well as in the body. That insulin corrects the adrenergic effects of fear is shown by Funkenstein *et al.'s* report (1950) that the fall of blood pressure on injection of mecholyl is intensified after a course of insulin treatment, while the blood pressure rise after an injection of adrenaline is decreased. Soon the patient begins to feel better, has more appetite, and is able to deal with his problems more effectively.

Electroshock also seems to induce a beneficial reaction to the autonomic effects of mental disturbance. Its primary action is a profound vasodilatation as a result of the intense motor stimulation. Since this is a brief but intense effect, there is a profound and lasting vasomotor reaction. Consequently, a course of electroshock treatments decreases the fall of blood pressure on injection of mecholyl and increases the rise of blood pressure on adrenaline injection (Funkenstein *et al.,* 1950). It has also been found that the sympathetic reaction to anoxia (measured by the increase in blood sugar) was more pronounced after twenty-four electroshock treatments than after ten. However, after a rest of thirty days, sympathetic reactivity had decreased again, in some cases below the initial level (Gellhorn, 1953). This illustrates the continuous homeostatic checks and balances, which may allow a temporary overbalance in one or the other direction but eventually establish a working equilibrium. The effect of electroshock and the organismic reaction to it, which engages the action circuit maximally and stimulates both branches of the autonomic nervous system, might explain why electroshock is most beneficial in depression where total central nervous activity is severely inhibited.

Funkenstein *et al.* (1948) found that electroshock increases the anxiety induced by adrenaline but reduces the agitation produced by mecholyl. In fact, the outcome of electroshock treatment could be predicted according to the patient's blood pressure reaction to a mecholyl injection. If there was a profound long-lasting fall in blood pressure, the patient improved in more than 90 percent of the cases. If there was a moderate fall which quickly returned to base or remained above the base line, there was no improvement. Apparently, it is the secondary reaction to electroshock rather than the primary effect that restores the organismic balance.

Indirect therapy. Since a direct attack on the stressor or an attempt to reduce the effects of stress either produces new undesirable effects or delays and intensifies the original effects, and since a reinforcement of the vital reaction eliminates the self-limiting feature of the normal reaction, it would seem preferable to use a type of therapy that seeks to establish a new and more favorable balance.

A disturbance never has a purely physical or purely psychological effect. We may not always know that there is danger of a serious disturbance (e.g., when exposed to radioactive fall-out or to infection), but we always feel its effects. Any severe stress affects the whole bodily economy, and with it the brain. There will be a profound general malaise and there may also be changes in perception, feelings, or emotions. This subjective experience of a disturbance has been woefully neglected in diagnosis. Convinced that self-observation is not reliable, we have refused to avail ourselves of this information even when it may give us more knowledge than the observation of physical symptoms. When a man complains of pain and no lesion can be found, we too easily assume that his pain must be the expression of a psychological difficulty. It has almost become the fashion to attribute to psychological causes whatever cannot be diagnosed with precision by objective methods. The recent investigations of drugs like d-lysergic acid or mescaline, which produce experiences resembling those in psychosis, promise a new understanding that changes in perception, feelings, and emotion can be the result of definite physical disturbances.

The vital response to a given type of disturbance very rarely represents an equal and opposite reaction. In this respect a living system seems to react rather differently from a nonliving system. We have tried to show that mild stress seems to induce a vigorous organismic reaction that may actually improve the bodily economy. Moderate and gradual exposure to cold produces acclimatization that makes it possible to be comfortable at temperatures that were extremely uncomfortable before. As stress increases, the vital reaction cannot keep step and the effects of stress begin to dominate. To increase the organismic resistance, it would seem desirable to apply a very mild stress briefly and repeatedly so as to stimulate an intense vital reaction that can counteract even a severe stress of a similar type. In this way we employ the natural organismic reaction without bypassing its self-limiting mechanisms. Vaccination against small pox or infantile paralysis makes use of this principle. The micro-organisms are killed before the vaccine is injected and so they are prevented from multiplying and flooding the body with toxic substances. Yet the injected toxins provide a stimulus for the vital reaction which can develop such an impetus that permanent immunity or immunity for long periods will result.

This principle of stimulating a healing reaction with small doses is also used in desensitizing against various allergic reactions; apparently, it is possible to induce a healing reaction by applying a very mild stress of the same type even at a time when the effect of stress is still continuing. Whether the allergy is produced by injection of a foreign protein into the blood stream or the repeated exposure to various usually harmless substances (flour, pollen, hair, etc.), it is possible to induce a vital reaction to their *effects* by small doses of the same substance. These effects (e.g., local vasodilatation, asthma, conjunctivitis, etc.) can also be counteracted by giving glucocorticoids. But these have to be continued for as long as relief is desired while successful desensitization protects the patient for a long time.

The organismic reaction may even be exhausted in response to a particular disturbance, yet may be stimulated again by a related stress. Thus Talbot *et al.* (1952) mention that infantile eczema often improves during pneumonia and other acute diseases. They suggest that the organismic reaction may become fatigued during chronic stress and need an acute stimulus to revive. It is interesting that Hahnemann recognized this phenomenon in the early years of the last century and put it to use by employing minimal doses of drugs that have a similar effect as the disease.

Psychotherapy is necessarily concerned with the person as a whole, although some theorists, in accord with medical usage, have attempted to deal directly with the original cause of the disturbance. Even when this is done (e.g., by tracing the traumatic event to childhood), it is generally recognized that the effects of early trauma must be counteracted before a cure is possible. In the process of working through the patient's associations, the therapist makes it possible for his patient to see and appraise his own reactions objectively and so, in the end, to correct them.

When the psychotherapist hopes to supplement the vital reaction directly, as does Wolpe (1952), for instance, who attempts to replace neurotic fear by anger, he helps the patient to see a difficult situation as something to conquer rather than as something to avoid. Here also, the real aim is to change the patient's outlook and alter his final equilibrium. Thus psychotherapy always tries to establish a new and favorable balance, though some systems do so deliberately, supported by theory, while others do it incidentally. This effect of psychotherapy is probably responsible for the physical improvement so often noticed. This does not mean that the physical symptoms were necessarily caused by a psychological disturbance. Many a patient who has been shuttled back and forth between internist and psychotherapist because no palpable organic disturbance could be found, has improved on psychotherapy only to die of cancer.

Scientific as well as cultural trends seem to progress by way of thesis and antithesis to eventual synthesis. One generation of scientists may see every disturbance as organic, the next may overemphasize psychological causes; let us hope that the synthesis to come will bring a realization that disturbances of every kind affect the total man who suffers their effects and reacts to them. Eventually, we may realize that both somatic and psychological effects have to be known if a remedy is to be found that will bring about a new balance, i.e., health.

Part III. THE ROLE OF EMOTION IN PERSONALITY

9. EMOTION AND PERSONALITY ORGANIZATION

We have seen that different emotions produce different physiological changes. Anger and rage are accompanied by massive motor innervation and the secretion of noradrenaline, and eventually lead to muscular incoordination. Love and affection have mild cholinergic effects that lead to a sense of well-being and heightened efficiency. Fear, on the other hand, excites adrenergic pathways. If it is mild and the threat can be avoided, the harmful effects of temporary sympathetic excitation are negligible. But if fear is pronounced or chronic, the intense sympathetic excitation may seriously impede normal physiological functioning.

Emotions related to fear will have similar effects. Sadness, sorrow, and depression show a heightened level of sympathetic activity without the usual parasympathetic reaction (Funkenstein *et al.*, 1949). As a result, the action circuit and perhaps even the estimative system seem to be severely inhibited, which accounts for the indifference and "retardation" so characteristic of such states. Impatience and irritability, on the other hand, seem to be the result of increased excitability of the estimative system, which can be brought about by a number of factors, among which the habit of reacting impulsively, without deliberation, surely plays a role.

EMOTIONAL EXPRESSION

Normally, every emotion has some effect on our functioning. Normally also, there is a continual stream of feelings and emotions, aroused by our dealings with the world around us. Perception ordinarily leads to an immediate intuitive appraisal, and that automatically results in emotion. Once emotion is roused, it leads spontaneously to emotional expression, for it activates the autonomic as well as the somatic nervous system. The emotional impulse urges not only to action but also to a preoccupation with the situation that has precipitated the emotion. It coerces attention and so reinforces the emotion. We have seen that there is a neural mechanism that mediates appraisal and emotion and a circuit that allows for attention, self-initiated imagination, and self-directed action.

Whatever the emotion, once it is ended, the organismic balance will be restored, though not completely. We have seen that every appraisal brings with it an expectation that the object of our emotion will remain constant in its effect upon us. Every new appraisal of a similar situation builds upon earlier expectations, eventually creating an enduring emotional attitude. Though the physiological changes may not be large on each occasion, they will have a cumulative effect if the emotion occurs repeatedly, for the disposition to such changes seems to be preserved and registered as autonomic motor engrams in the prefrontal association areas. When an emotion recurs persistently, the emotional attitude that develops from it will eventually stand out among others and will dominate the person's outlook as well as influence his bodily functioning.

Posture and muscular tension. The most obvious example of such cumulative changes in bodily functioning is the habit of muscular tension and posture as it develops in different individuals. A small child is extraordinarily relaxed, almost as much as a kitten. Like a kitten, it can fall dozens of times a day without getting hurt. But the human adult is far from having the flexibility and relaxed grace of a full-grown cat. There are far-reaching individual differences in the degree of muscular tension. The anxiety neurotic is almost rigid, particularly when he attempts to relax (Jacobson, 1940). This increased muscular tension seems to be a direct effect of increased adrenaline secretion. According to Brown *et al.* (1948), adrenaline increases muscular tension by retarding the spread of excitation along the muscle fibers, which would explain the neurotic's poor performance as well as his chronic fatigue. In contrast, the easygoing, jolly extrovert moves with a confident grace that reveals effortless coordination. Posture is similarly revealing. The uneasy slouch of the girl who is too tall, and the squared shoulders, cocky walk, and roving eye of the Don Juan who is determined to conquer, speak louder than their possessors know.

Such habits of tension and posture are the result of attitudes an individual develops toward things and people around him, of his habitual readiness for particular actions. The girl who thinks she is too tall is afraid of seeming so to others. She wants to be short and inconspicuous and tries to shrink as much as she can. The cocky conqueror sees every girl as a challenge that promises a victory, and proclaims his confidence in the very way he walks. The girl knows only that she is ill at ease in a group but does not realize that this stems from her fear of being thought too tall. The young man knows well enough that every pretty girl attracts him and that he wants to pursue her, but may not know that the beckoning victory attracts him more than the girl. In both cases, there is a habitual intuitive estimate and a habitual emotion with all the physical changes that go with it, even though the person may be unaware that he shows it so plainly. The attitude stemming from this emotion has become part and parcel of the individual's outlook on life, his self-concept and self-ideal. With repetition, the physiological effects of his habitual emotion have become cumulative, and gradually they leave their mark on face, posture, and movements.

This habitual muscular tension, as shown in posture and facial expression, can be reduced but not abolished by general muscular relaxation —merely training a man in relaxation methods will not affect his habitual muscular tension in daily activities (see Jacobson, 1929). Such habitual tension can be retrained, however, by methods designed to reduce muscular tension during movements and train smooth muscular coordination (cf. Lee, 1946; Alexander, 1910; for Hatha-Yoga techniques, see Behanan, 1937). It is significant that emotional explosions may occur as such training progresses, betraying buried anxieties no longer held in check. Both Jacobson and Fenichel (1945) have noted that a reduction of tension by relaxation techniques is often resisted by neurotics who feel helpless without such vital armor. Conversely, when anxiety is relieved by psychotherapy, muscular tension lessens, though training in relaxation and coordination is required before optimal coordination is achieved. Old habits of muscular tension, once vitally connected with anxiety, have become so fixed that they are retained even though the anxiety has disappeared.

That such muscular tensions are actively maintained by attitudes of fear is shown dramatically in prefrontal lobotomy. As soon as the incision is completed, there is a noticeable reduction in muscular tension. The patient loses his pallor, his drawn look, and his skin becomes pink and smooth. Since this operation interrupts the projection tracts from the thalamus to the frontal association areas, the automatic revival of motor memories is prevented and the cumulative effects of emotion are abolished.

Obviously, the effects of emotion on the body explain many symptoms found in various psychiatric disorders. The neurotic patient who is continually fatigued, whose work capacity is seriously impaired, and who suffers from hypertension and palpitation demonstrates the physical effects of chronic fear. The psychotic who is aggressive and destructive seems to act out the emotions induced by his hallucinations or delusions. The "effort syndrome" so prevalent during the Second World War and many cases of gastric ulcer or hypertension are the physical expression of emotion, even if that emotion is not recognized. It would be tempting to follow the physiological effects of emotion into the realm of pathology. Psychosomatic disorders, neuroses, and psychoses exhibit these effects to varying degrees. However, we shall limit ourselves to the effects of emotion on normal personality.

EMOTIONAL DISTURBANCE

As a rule, emotions help man or animal in the pursuit of their goals. Negative emotions and feelings help in selecting goals, in keeping away from what we dislike or distrust, while positive emotions supply the attraction to things beneficial that results in effortless action. Instincts themselves must contain an emotional component, a tending toward the appropriate object, before they can effectively move the living being to instinctive action.

When the environment is not altogether favorable, contending emotions aid in striving for something that is not immediately available, in holding onto it and defending it against all comers, in overcoming difficulty and danger or, if that is not possible, in escaping from it. While impulse emotions make it possible to reach out toward anything that is easily accessible, contending emotions urge us to obtain what is needed in spite of obstacles and to retain possession of it (see Vol. I, Ch. 10). These two modalities of emotion (impulse and urge) seem to constitute the basic tendency of man or animal toward the *possession* of things needed to develop and maintain himself or his species, and the tendency to *establish himself* in their possession. His emotions, expressed in these basic tendencies, seem to organize the living being in his relations to the world around him (cf. Gasson, 1954).

These spontaneous tendencies are sufficient for the animal to reach the perfection his hereditary endowment and his environment permit, but the human being needs more than that. For one thing, emotion itself may disturb a man's deliberate purposes. Animals are guided by their emotions to what is beneficial and kept away from what is harmful, as long as they are in their natural environment. Barring catastrophes, an animal's emotional tendencies will urge him to appropriate action. But in a human environment, conditions may change rapidly, often at the whim of parent, teacher, manager, or experimenter. Such changes may arouse intense emotions, making effective action difficult. And continual emotional upsets, as we have seen, leave a residue that may culminate in attitudes of timidity or anxiety.

Sudden emotion. Emotion disturbs goal-directed action whenever it captures and monopolizes our attention while we are otherwise occupied. This happens most easily when there is a sudden threat that breaks into the customary routine. Hamilton (1917), for instance, devised an ingenious experiment. His subjects learned to find their way out of a locked room through one of four doors. They had to discover a simple principle that would help them to find the open door; the door found open last time was always locked in the next trial. When the men were unexpectedly doused with cold water, or the animals were given a strong electric shock, they tried frantically every door, often over and over, and seemed to have forgotten everything they had learned before. Hamilton (and others after him) called this phenomenon "regression" and assumed that emotion reduces the organism to a lower level of functioning.

But we must not forget that the situation had radically changed as soon as dousing or electric shock was introduced. Before, the men had learned the problem for a particular purpose, namely to find their way out according to some principle; the animals had learned a way to food. At the moment when cold water or an electric shock surprised them, they were no longer looking for the old solution, they were now trying to escape from a situation that had suddenly become unpredictable. What before had been a game, now became menacing. For all they knew, something might have gone wrong and they might be in serious danger. And if one mechanism can go wrong, so may another; now any door might be unpredictably open or locked and the only thing to do was to try them all and try them repeatedly.

To call this change in behavior "regression" and to say that emotion makes the human being regress to the level found normally in lower animals, as Hamilton does, is a poor way of saying that the basic emotional urges are the same in man and animal. If human beings are taken unawares, they will show emotional and not rational behavior. Sudden danger will result in immediate intuitive appraisal which cannot stop for rational deliberation. At the same time, it is possible to train even such immediate appraisal, as shown in the lightning-quick actions of a skilled driver who avoids a collision before he has a chance to think about it.

Recently, Reece (1954) has shown experimentally that emotional upset does not result in disorganization when the situation is so arranged that the person can learn a means of escape. He let his subjects learn pairs of nonsense syllables appearing on a memory drum. Then he projected these syllable pairs by means of an electronically controlled tachistoscope and recorded the time each individual took to recognize them. Painful electric shock was given with certain syllables. One group could end the shock as soon as they called out the syllables, while another group had to suffer the shock whether they called out the syllables or not. All groups recognized these syllables more quickly after the experiment, but the group that could end the shock by recognition took a significantly shorter time than the non-shock control group. In contrast, the group that had to suffer shock throughout exposure of the shock syllables showed but slight improvement. During the following interview it became clear that those who could end the shock interpreted it as punishment for errors, failure to respond, etc., while the others simply saw it as unwelcome interference. Few subjects admitted "anxiety," but their startle and pain were evident. They showed sudden, profuse perspiration, and mispronounced syllables; there was much shuffling of feet, clearing of throat, and voice tremor. Apparently, the emotional upset was quite as pronounced as that of Hamilton's subjects, yet those who could end the shock, unmistakably learned to recognize the syllables more quickly than the control group.

We can conclude that emotion does not necessarily reduce the person to a lower level of function or interfere with his performance. Emotion interferes when it suddenly diverts his attention from his task. But when it is part and parcel of the learning situation, it neither distracts nor disorganizes. A good example is the fear before a public performance, whether that is a sermon to be preached, a lecture to be given, or a role to be played. If this fear is given in to, stage fright may completely disrupt the performance. But if a man takes the task before him as a welcome challenge for his powers, he may give an excellent performance, far better than if it had left him indifferent.

When emotion is irrelevant to the task, it will distract attention and disturb performance, whether the diversion is pleasant or unpleasant. The child who is supposed to practice his piano lesson and sees his playmates outside the window will have a hard time keeping at his task. The student who cannot concentrate on his examination questions because he is worried about a quarrel he had with his girl will find it difficult to write a good paper.

Excessive emotion. Emotion can become disturbing even when it prompts to actions that are rationally desirable. Longing for home or striving for advancement can become so insistent that it finally overrides all reflection. Anger can become so blinding that it leads to an action later bitterly regretted. Fear can become so overpowering that it paralyzes. Courage can become foolhardiness and may end in despair if the danger proves greater than our strength. Contending emotions always carry the danger of excess within them; and excessive emotion is harmful, for it excludes the awareness of all other legitimate interests as long as the emotion lasts. In addition, the excessive stimulation of the autonomic nervous system taxes bodily resources and may seriously interfere with physiological functioning.

Chronic emotion. Not only excessive but chronic emotions disturb human functioning. Emotional attitudes bias the appraisal of new situations and intensify our reaction to them. In the nature of the case, this is particularly harmful with negative emotions because they make a constructive approach difficult and have undesirable physiological effects. Chronic fear produces the host of symptoms we find in neurosis. Chronic worry and chronic resentment have other, no less damaging effects. Chronic sadness has the same effects as found in reactive depression.

EMOTION AND CONFLICT

Emotion is disturbing when it breaks in on another emotion and on the action that flows from it. This happens particularly when the situation arouses not only desire but fear. The boy about to be introduced to his admired football hero is afraid that he will not find the right word. He blushes and stammers and is further mortified by his embarrassment. The young man wanting to declare his love is speechless for fear that he will be refused and is even more crushed by his emotional disturbance. In these cases, an action is urged by intense emotion but disturbed or prevented by fear of personal ineptness or unworthiness. Thus the appraisal that aroused the emotion and led to approach is replaced by another which arouses fear and makes for withdrawal. As long as a person oscillates between the appraisals, there will be opposing emotions with opposing action tendencies. This clash constitutes an emotional conflict.

To experience a conflict, both tendencies must act for some time. An action tendency that delays but does not prevent reaching a coveted goal will not produce a conflict. Likewise, a goal that is resolutely relinquished when an opposite tendency interferes will cease to be an occasion for conflict. Instinctual tendencies do not result in conflict if one is satisfied after the other. A man who is both hungry and sleepy may be uncertain whether to eat first or to sleep before eating, but whichever he chooses, both desires will be satisfied in the end: there is no conflict. Similarly, renouncing one tendency, even an instinctive tendency, does not bring conflict. A vegetarian who out of conviction has decided against eating meat has no conflict. Meat simply ceases to be something for which he will strive.

There is a conflict, however, when there are two contrary action tendencies that continue, and one of them, at least, is not instinctive. Instinctive action tendencies are always positive, but emotional and rational action tendencies may be positive or negative, urging to approach or withdrawal. When such a negative action tendency interferes with a positive tendency, there is conflict. In the case of the timid football enthusiast and the bashful lover, one emotion interferes with another, for one is urging withdrawal when the other prepares for approach.

Conflict in animals. When conflict occurs in animals, it is always a negative emotion that interferes with a positive (emotional or instinctive) tendency toward the goal. What creates the conflict is the fact that the same thing (e.g., food) that is wanted has also become an object of fear. This, however, is a rare occurrence. In their natural habitat, what attracts animals is also good for them and what frightens them is really harmful. Only in psychological laboratories do animals experience one and the same thing sometimes as beneficial, sometimes as harmful. We make a lever attractive for the animal by having it deliver food pellets whenever it is pressed, and when rat and cat begin pressing it avidly whenever they are hungry, we frighten them by airblast or electric shock every time they touch the lever until their fear interferes with their desire for food. If the animal has no other source of food, it will approach the lever repeatedly, only to get another shock. Eventually, fear will become chronic and the animal will appear "neurotic"; yet the cause of the neurosis is not the conflict but the unremitting fear.

Another conflict that is thought to produce neurosis is the so-called "perceptual conflict." One signal announces shock and a different signal stands for no-shock. When the animal has learned to distinguish them, the signals are gradually made more and more similar until the animal can no longer tell them apart. Clearly, the conflict is not between "perceptual tendencies" but between opposing action tendencies. The signal for shock arouses fear, the signal for no-shock promises safety and allows approach. As the two signals become more and more similar, the animal finds it more and more difficult to know when it is safe to approach and when not. Eventually, every signal is appraised as threatening and the continued apprehension produces neurotic behavior. In every case, so-called "animal neurosis" seems to be the product of chronic fear and not of conflict (cf. Liddell, 1956).

Human conflict. We have argued that there is conflict whenever a tendency to approach is counteracted by a tendency to withdraw. In man, the interfering action tendency may be either a negative emotion or a deliberate decision to inhibit action, prompted by a reflective choice. When something that is appraised as attractive is not acceptable to the person, there may be a conflict. This conflict is cut short as soon as a man makes a clear decision to turn away from the attraction and succeeds in following his choice wholeheartedly. Conflict is experienced only when the proscribed attraction remains in the center of attention so that there is continued longing, half-hearted restraint, and partial indulgence.

If a diabetic wants to diet but cannot keep his mind from the thought of sweets, he will experience a real conflict; he will be torn between his desire for candy and the fear of the effect of his indulgence. The poignancy of his conflict comes from the steady vigilance that is necessary to keep his resolution while the sweets he craves are as tempting as ever. Perhaps the old distinction between sin and temptation could be revived in this context: if the conflict is really resolved, there may still be temptation (sweets do remain appealing), but there will be no "sin" because the attraction is resolutely put out of mind. But an unresolved conflict means a painful alternation between attraction and repulsion, a hesitant giving in and a reluctant refusal. Conflict breeds disturbance: there is the craving that is never satisfied and never conquered; the anger and rebellion against the prohibition; fear of the consequences should it be transgressed; shame, remorse, and eventually self-contempt if the desire is fitfully indulged. While it is safe to say that fear is always involved in conflict, it is by no means the only emotion that springs from it.

Neither emotion nor instinctual impulse leads a human being to action directly and immediately. Both these action tendencies can either be restrained or indulged. Human conflict is rarely a simple clash between two emotional action tendencies. Rather, a negative emotion (fear, embarrassment) may interfere with an action decided on reflectively, or an emotional tendency may be restrained deliberately. Either emotion urges withdrawal in spite of a deliberate decision to approach, or emotion urges approach though it is reasonable or advisable to withdraw. (For a discussion of deliberate control of action, see Ch. 6.)

Freud's view on conflict. For Freud also, conflict consisted essentially of the clash between a tendency to approach and a tendency to withdraw (cathexis and countercathexis), but in his theory the tendency to approach is initiated by the id while the tendency to withdraw stems from the ego (forced by anxiety, i.e., fear of reality and of the superego). Freud, too, believed that instinctual impulses do not lead to action directly and immediately but can be restrained by the ego. To resolve the conflict, there must be sublimation instead of countercathexis. According to Fenichel, all successful defenses against id impulses come under the heading of sublimation:

> Sublimation is to be differentiated from defenses that use countercathexes; sublimated impulses find their outlet, though drained via an artificial route, whereas the others do not. In sublimation, the original impulse vanishes because its energy is withdrawn in favor of the cathexis of its substitute. In the other defenses the libido of the original impulse is held in check by a high countercathexis. (1945, p. 141)

Apparently, sublimation substitutes an acceptable aim and thus allows discharge of libidinal energy instead of blocking it. If the conflict is not resolved by sublimation, the necessary countercathexis will drain the organism's energy resources. Here again, we meet a metaphor instead of being given an explanation of how sublimation and countercathexis draw upon energy, either physical or psychological.

At any rate, since sublimation represents the successful resolution of a conflict which can be achieved only when the original impulse is not repressed, the conflict here must have been conscious. For Freud, it would become unconscious only when it cannot be resolved, i.e., sublimated. Thus psychoanalysis makes a distinction between a conflict that is immediately resolved by redirecting the energy discharge and has no harmful effects, and a clash between an instinctual tendency and ego defenses, which results in the repression of the original impulse and has harmful effects. In Freud's conception, the original impulse remains active because it is an instinct, and the repression is maintained by anxiety because the danger from the instinct persists. Yet we have shown that the "return of the repressed" in neurosis does not mean a breakthrough of the instinct but an eruption of anxiety, and it is not the repressed instinct that is harmful but the continuous anxiety (see Vol. I, Ch. 12, Psychoanalytic Theory of Anxiety).

From our point of view, unresolved conflict is harmful even when it is conscious, because the two opposing tendencies are accompanied or constituted by emotions which may easily crystallize into dominant attitudes. When fear is involved, it may easily become chronic and lead to neurosis. The physiological effects of the emotions stemming from the conflict will continue as long as the conflict lasts and will make it increasingly difficult to come to a decision.

Conflict between two rational alternatives. The situation is similar when two rational desires are incompatible, for instance, when a student cannot decide whether he should become a physicist or a physician. The choice may incline first toward one and then toward the other alternative so that he becomes worried and anxious. Or his choice may be made unwillingly and later regretted. The desire that is denied is kept alive by continually picturing the advantages of the other choice. A man may also want something without wanting to use the means to achieve it (like wanting achievement without self-discipline) or without wanting the consequences (like wanting promotion without the responsibility that goes with it).

Finally, there may be a conflict because two reasonable goals cannot both be chosen at the same time. A man can either catch up with a backlog of work or he can devote the time to his family, but he cannot do both. When two reasonable alternatives clash, the conflict is difficult to resolve because each alternative is attractive in some way and there is good reason for choosing it. Moreover, there are penalties if either alternative is rejected. When a man's work piles up, there is that much more for him to do. When he does not spend some time with his wife and children, he deprives them and himself of rightful joys. The fact that he has had to make the same impossible choice in the past and has had to suffer the penalties intensifies his conflict. The choice is so painful because each alternative is desirable and also undesirable. Whatever he does, he is bound to regret it, even if he should decide that the choice is too much for him and he goes on a binge instead.

To sum up: Conflict is felt when a tendency to approach an object clashes with a tendency to withdraw from it. In man, both these action tendencies may be deliberate, or voluntary, though at least one of them must be accompanied by considerable emotion before there can be a conflict. In a conflict a choice is either delayed or it is made and revoked so that the two opposing tendencies are sustained. As Gasson puts it: "When the conflict is conscious it will be found to be the result either of an inability to choose because the person is not willing to sacrifice the alternative goal, or it is the consequence of making a choice unwillingly and wanting the alternative, or finally, of regretting an irrevocable or necessary decision" (1954, p. 204). In such conflicts the emotions are those aroused by the original goal as well as those stemming from the restraint. A conscious conflict is resolved as soon as a choice is made and the person abides by it, so that the opposing desire disappears because its object is no longer in the center of attention. A conflict can become unconscious if it is decided in favor of the emotional attraction. If that happens, the prohibition is not discarded but merely disregarded and this leads to disharmony and frustration.

Control of emotion. There is no doubt that conflict disrupts personality organization unless it is speedily resolved. Since there is emotion with every conflict—urging us to some actions we find undesirable or holding us back from others that are useful or necessary—control of emotion becomes imperative if effective personality organization is to be achieved. Such control does not mean merely that emotions should be reduced or restrained, or that the actions to which they urge should be omitted. More to the point, emotions should be controlled in such a way that they help rather than hinder personality organization.

Emotions may distract us: the boy who wants to train for a profession cannot afford to spend a great deal of time on hobbies, however pleasant or interesting they may be. Emotions may actively hinder us: a boy who is painfully shy will find it exceedingly difficult to become a public speaker or concert pianist, no matter how talented he may be. In addition, the sheer physical effect of emotion can turn liking into craving, love into bondage, fear into cowardice, and make reasonable action all but impossible.

Reasonable human conduct demands that we reduce the intensity of pleasant emotions that distract us from a chosen goal and overcome the negative emotions that prevent us from following it effectively.

One way of reducing the intensity of pleasant emotions is to increase the desirability of the goal with which they compete. This can be done by thinking about the goal, dwelling on its value, its undoubted merits, and further, by planning the steps to take for reaching it and rehearsing them in imagination. At the same time, the emotional attraction of things that tempt us can be reduced by doing without them voluntarily for a time, even when they do not interfere with our goal directly.

We have seen that a habit of indulgence is easily established (see Vol. I, Ch. 9, Attitude and Habit). With the first indulgence comes the temptation to repeat it until it becomes a habit. This applies to pleasures of taste and touch even more than to the delights of sight, sound, and movement. Concentration on any of them is almost certain to produce a craving that is difficult to restrain. There are not only drink and drug addicts but also people who crave sweets or rich food; there are TV and movie addicts, rock 'n' roll and dance addicts. When the physical effects of indulgence intensify the craving, as happens with alcohol, opium, and other drugs, or with tobacco, it is seldom possible to taper off or voluntarily restrict their use. At the same time, an uncontrolled craving may lead to actions that not only interfere with personality organization but that lead to downright disorganization. The plight of drug addicts is an eloquent warning. Once craving has reached such proportions, the only remedy is to renounce indulgence altogether.

To overcome negative emotions that hinder reasonable action (fear, shyness, embarrassment) it is necessary as with pleasant emotions to increase the desirability of our goal by dwelling on it in thought and imagination. Next, we must plan ways and means of doing what we have decided on. Once we have made our decision, we should let ourselves become interested in what we are planning and in the best ways of bringing it about, without letting our thoughts stray to possible difficulties or even dangers. If our purpose is strong enough, we may even picture the worst that could happen and ask ourselves whether we would not gladly suffer what is in store for us for the sake of doing what we set out to do. Thinking and planning what is to be done will intensify the wish to do it, and realizing that whatever we fear is not the worst that could befall us will give us the courage to risk it.

Obviously, such steps are helpful only when emotions have not yet become firm attitudes that have forced a man's action into iron grooves of habit. Obviously also, such control of emotion implies a worth-while self-ideal to provide a focus for a man's striving. A man may develop habits of right action (they used to be called virtues) or he may fall into habits of indulgence (formerly called vices). Since the attachment to things pleasurable is strong and the pull of contending emotions difficult to resist, habits of reasonable action can be formed only if that emotional urge is reduced. Giving in to the attraction of the merely pleasurable, to emotions of fear and anger, to the desire for power or money, will develop habits of action that intensify this pull. Such habits of indulgence will inevitably distract a man from achieving what truly enhances his human nature. They will be obstacles to achieving his true self-ideal, the perfection he could achieve given his capacities and opportunities.

Pleasures or emotional satisfactions need not be rooted out ruthlessly. They are a legitimate part of the joy of living. Unfortunately, they attract us so strongly that they may easily crowd out our striving for reasonable goals. Their lure is far greater than their value for a well-ordered, happy, and effective human personality. Emotional control demands a direction of attention toward spiritual values and a turning away from the merely pleasurable things of life, at least for a time, until their hold is loosened. Emotional control means both a turning toward what is truly lovable from a human point of view and a turning away from things that exert too strong a pull.

EMOTION AND THE SELF-IDEAL

The very fact that a man can withstand an emotional attraction or act in spite of fear raises the question of why he should want to do it. Both the psychological and physiological effects of emotion combine to make any action prompted by it easy and pleasant, any resistance to it intensely unpleasant.

The human infant, like the animal, gives in to his emotions without hindrance. Indeed, his emotions urge him to reach out toward people and things around him and thus make it possible for him to grow and develop. Freud seems to have had this primitive organization in mind when he suggested that the pleasure principle is the prime regulator of infant activity. It seems to us, however, that this first organization is not an organization of *instincts,* as Freud proposed, but a product of the inherent tendencies we experience as *emotions,* which combine in the basic *tendency to possession and self-establishment.*

In the animal, this organization is final. Throughout life, the animal is determined by emotional attraction and repulsion. Such emotions, of course, include the affection for any human being that feeds an animal or cares for him. The fear of his master's anger (not just the fear of being punished) is a real and powerful motive for a dog. But only on Freud's supposition that the later organization of human activities is also forced by emotion (fear of punishment and loss of love, internalized in the superego) could it be considered likely that dogs have a superego, and at the same time, that the superego represents the promptings of conscience.

In the human being, emotions do not remain the only action tendencies, though they always continue to influence him. We have seen (Vol. I, Ch. 11; Vol. II, Ch. 6) that human beings are motivated by an appraisal that is both a sense judgment and an intellectual or reflective judgment. The final decision for action is a choice that either implements the original emotion or goes against it. In man, the choice of goal-directed action is essentially a rational wanting, an inclination toward what is reflectively appraised as good (pleasurable, useful, or valuable). These rational action tendencies organize the human personality under the guidance of the self-ideal.

Freud's theory of the superego. To say, as is often done, that even rational actions and moral conduct have unconscious irrational motives is neither good sense nor good science unless the assertion can be proved. The proof given is usually another assertion, that in the last analysis our sense of oughtness, our conscience, is the product of an early fear of punishment and loss of love. According to Freud, the child identifies with the punishing father and introjects him as the "superego." The identification represents a desexualization, a "defusion" of the instinct, which releases the aggression formerly bound by the libido and turns it against the ego. This accounts for the harshness of the superego and its imperious demands. For orthodox psychoanalysts (and the Yale school of theorists) the superego represents an internalization of parental punishment to this day, as appears from Fenichel's comment that "the outstanding identification takes place with that parent who was regarded as the source of the decisive frustrations" (1945, p. 104).

Accordingly, this internalization of parental figures develops into the child's later conceptions of right and wrong. Thus the superego is responsible for self-observation, self-judgment, and self-punishment. Before we discuss whether human conscience is adequately explained by postulating the formation of a superego, let us examine the way in which this process, in Freud's opinion, is brought about.

The process of identification. Apparently, the superego is formed by identification with the father; Freud compares this identification with an "oral-cannibalistic incorporation" (1933, p. 90). For Freud, identification is a process in which the ego becomes identical with the object. This identity is established by introjection and incorporation, even though the introjected object may secondarily assume an identity of its own in the form of an autonomous superego. Freud's statement that the ego, in identifying with the object, "recommends itself to the id in the place of the object" (1933, p. 108) leaves no doubt that identification means assuming the identity of the object.

But postulating identification and introjection is not enough. There must be a reason for identification and some means by which introjection can be brought about. According to Freud, the little boy resorts to identification when he fears losing his father's love, and also when he takes his father as a model, when he "would like to be like him, and take his place everywhere" (1921, p. 61). But alas, the father turns into a feared rival as soon as the son wants to take his place with the mother. Thus identification is always ambivalent; it includes hate as well as love. Since the father is loved as well as feared, the father image introjected as the ego-ideal or superego draws the libido to itself while the defused aggression is turned against the ego. In infancy, the libido is centered on the ego; now it is displaced to the superego. Thus the ideal incorporated in the superego reigns by virtue of the love it draws from the id as much as by dint of the aggression that forces the ego to follow its behests.

Freud's model of energetics, of course, required such an account. In his system, it is not possible to have an ego-ideal that commands devotion as it commands obedience, without some process of energy exchange. Identification and internalization seemed to offer a method by which energy could be trapped and concentrated into an autonomous system. Thus a superego could be formed that would "rage" against the ego but command love from the id and so have the power to force its commands on both ego and id. Once more, Freud uses the analogy with a system of energetics to explain the intricacies of human motivation. His analogy is picturesque, even engaging, but it does not explain how a human being can "introject," how he can establish a self-ideal and then be motivated by it.

Formation of the self-ideal. When we leave Freud's engaging metaphor and go back to his descriptions, we find that the child takes the parents as his model because in knowing and loving them he discovers that they are admirable, that they have qualities the child lacks. For Freud, emotion (whether love or aggression) is merely the affect charge of instinctual energy and cannot provide the power for action. For us, emotion is a tendency that prepares for and urges to action.

Taking somebody as a model, as an ideal toward which to aim, presupposes a realization that it is a good thing to be like the model and a bad thing not to be like it. The judgment that this is good and ought to be followed is immediate and intuitive but also conceptual in nature, for it implies a comparison between the child as he is now and as he wants to be. As a sense judgment, it leads to emotion. As a conceptual judgment, it is an appraisal that being like father and mother is worth while, and not, that it is pleasurable or useful. The child realizes that father and mother are big, strong, and kind, and thinks them altogether perfect. He admires them and wants to become like them. Since love and admiration, like every other emotion, include an impulse to action, the child will be eager to do what is necessary to become like the ideal he admires. No separate process of identification or introjection is needed to explain his action; the sequence of perception, appraisal, emotion, and action is sufficient to explain how he can model himself on an ideal.

Reaching out to mother and father, admiring them, loving them, the child is guided in his choice of actions. If he had to depend on his own experience with the things around him to learn what is beneficial, what harmful, he might never reach adulthood. If parents were to guide him by demands and prohibitions alone, he might become a robot rather than a man who knows what is right and what wrong. But when the child sees what his parents do and recognizes them as lovable, admirable, he acknowledges their counsel as wise and obeys willingly, out of love for them. Over and above doing what they require, he will try to do what they do because he recognizes it as good. He models himself after those he loves and admires: first mother and father, older brother or sister, later teacher or older friend, even friends of the same age in whom he finds something he lacks and prizes. He learns by such emulation to discipline himself and to choose what is valuable rather than merely attractive.

With the beginning of reflection, and increasingly as he matures, he realizes that he must take a hand in achieving self-perfection by the deliberate choice of a goal in life. He takes stock of himself, tries to gain insight into his assets and liabilities, his strong and his weak points, and compares what he is with what he knows he ought to be. Thus a man's self-ideal is an index of his maturity, for it reveals his scale of values.

When a man begins to reflect on his goals in life, he will inevitably be influenced by the goals his community or culture considers worthy of effort. What is believed in his community, what is said by teachers and thinkers in his world about the nature of man, his purpose in life, his final destiny, is bound to shape his self-ideal. It may well be that later generations of social scientists will feel little pride in the fact that their science has helped to devalue the image of man and thus to distort the self-concept no less than the self-ideal of those who accept such dicta uncritically. There was a time when man hoped that he was "a little lower than the angels." But in recent decades "scientific" debunkers have instilled in him the conviction that he is a creature of lust, greed, and rapacity; worse than that, that he is a machine as blindly determined as the computer or the guided missile. Victor Frankl makes a telling point: "At first, [man] understood himself as a *creature,* formed *in the image of his creator,* God. Then came the machine age and soon he began to understand himself as a *creator* formed *in the image of his creation,* the machine" (1959, p. 46; Frankl's italics; my translation*). Even for a scientist, it surely is more alarming to have a man model his self-concept on his creation than to have him conclude that every creation must have a creator.

Frankl goes on to say that scientism (as exemplified by biologism, psychologism, sociologism) is a form of "homunculism" which in years past has

> held a trick mirror in front of man's eyes in which he saw his distorted image showing him that he is "nothing but" a reflex automaton, an apparatus of drives, a psychic mechanism, or a mere product of economic conditions. . . .

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* Frankl's original:

"Zuerst verstand er sich selbst als ein *Geschöpf,* und zwar *nach dem Ebenbild seines Schöpfers,* Gottes. Sodann kam das Maschinenzeitalter, und alsbald begann er sich selbst zu verstehen als *Schöpfer,* und zwar *nach dem Ebenbild seines Geschöpfes,* der Maschine.

> Homunculism can make history—it has made history. Think of the history of the past years; the view of man as nothing but a product of heredity and environment, or "blood and soil" as it was called, has let us slide into historical catastrophes. . . . As far as I can see, it is only one step, in strict logic, which leads from such a homunculus image of man into the gas chambers. (1959, p. 47; my translation [*])

This step can be taken because the victim has no value and hence the persecutor has no guilt.

The United States may have been preserved from the same bitter logic because the homunculus image of man has never become the official creed of the nation, no matter how many intellectuals may have subscribed to it. Perhaps we should be grateful that science and scientists have had less influence here than in Europe. Perhaps the American contempt of eggheads and ivory towers has helped to preserve us from the kind of scientism that both Hitler and Stalin made official overseas.

In every culture, religion is one of the forces molding the individual (if not the corporate) self-ideal. When that religion is not scientism but theism, it emphasizes the supremacy of God, who is the beginning and end of man's striving. According to the Judaeo-Christian tradition, man was created by God, who gave him his human nature and also the freedom either to perfect it or to frustrate it. The perfection of human nature lies in loving God and in doing His will.

The self-ideal formed by this tradition goes far beyond the self-ideal that could be established on the basis of self-interest or social cooperation. It implies devotion to a loving Father [†] and love for everything living because it has been created by God and is loved by Him. It requires detachment from the too pressing demands of man's appetites and desires and a fearless pursuit of what he knows is right, regardless of suffering, poverty, or contempt.

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[*] Frankl's original:

"Hatten doch die bisherigen grossen drei Homunkulismen—der Biologismus, der Psychologismus, der Soziologismus—ihm in einem Zerrspiegel ein Zerrbild seiner selbst vor Augen gehalten, demzufolge er eben 'nichts' war 'als' je nachdem ein Reflexautomat, ein Triebapparat, ein psychischer Mechanismus oder aber blosses Produkt von Produktionsverhältnissen. . . . Der Homunkulismus kann Geschichte machen—er hat bereits Geschichte gemacht. Denken wir doch bloss aus der Geschichte der letzten Jahre daran, wie die Auffassung des Menschen als nichts weiter denn ein Produkt von Erbe und Umwelt oder, wie man es damals nannte, 'Blut und Boden'—uns alle hat in historische Katastrophen hineinschlittern lassen. . . . Jedenfalls ist es meines Erachtens nur ein Schritt der Konsequenz, der von einem solchen homunkulistischen Menschenbild in die Gaskammern hineinführt."

[†] God may be a father image for the child who finds it easy to conceive of Him in the image of his father, whom he loves. To assume that God is "nothing but" an idealized father image is to imply that God cannot exist; and if people throughout the ages have believed that He does, they must suffer from an illusion. Many psychologists have been quick to seize on this explanation, not

The Christian, in addition, finds a living model of human perfection in Jesus Christ, whom he loves and admires and wants to follow. This following of Christ, as Scheler saw it, means

> true identification of essence and form of the person of Christ with oneself—not as mere knowledge or awareness, but as a becoming, reforming, ingrafting of one's own substance into the person of Christ . . . This "becoming" follows the act of absolute commitment and unification of one's own person to and with Christ, which must lead without fail to a redoing of his actions. (1923, p. 101; my translation *)

Thus love and admiration combine in the desire to *be* the Master as far as possible; to follow so faithfully, to be committed so completely, that the Master lives in his disciple. In Paul's words: "It is now no longer I that live but Christ lives in me" (Gal. 2.20). Such is the Christian's self-ideal.

The self-ideal compels not because it was the punishing father who served as its first model; not because there has been an instinctual defusion and now the released aggression is turned against the ego; this ideal like every ideal compels because it is seen as admirable, as right, and is passionately desired. As long as this appraisal and this love for the ideal last, it will command allegiance.

Function of the self-ideal. Human beings cannot give in to their appetites in the impulsive way animals do. Man cannot help reflecting on what he is doing and comparing it with what he ought to do. For animals, there is no "ought." Without thinking, they grow into the most perfect specimen of their kind permitted by their heredity and environment. But human

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realizing that it betrays a bias against religion which is as "unscientific" as any bias for religion. Without such a bias, they would have realized that the concept of God as Father is peculiar to the Judaeo-Christian tradition, and that even there it was slow to develop. Since belief in a supreme power has been universal in all cultures, its origin cannot have been a projected father image.

* Scheler's original:

". . . echte Wesens- und Gestaltidentifizierung der Person Christi mit der eigenen Person—nicht im Sinne blossen Wissens und 'Bewusstseins,' sondern eines Werdens, Umbildens, Einbildens der eigenen Personsubstanz in die Person Christi. . . . Dieses 'Werden' aber erfolgt durch den Akt der restlosen Einsetzung und Einssetzung der eigenen Person für und in Christus, die dann ohne weiteres auch Mit- und Nachvollzug seiner realen Akte mit sich führt."

beings must take thought, they must implement the blueprint that nature has laid down for them. That they do not necessarily develop a mature personality is demonstrated by the growing need for psychotherapy and other methods of correction and reeducation. That they can do so even though the environment or their heredity may be less than perfect is shown by the frequent success of such reeducation.

A man organizes his life by establishing some hierarchy of values from childhood on. As soon as the child's self-ideal begins to be formed, it becomes the touchstone of everything else he values. As the child grows into the man, he chooses his life work in accord with his self-ideal. He becomes a farmer, a worker, an artisan, a scientist, artist, or business man. He chooses his vocation because he values it (or the things it can bring him) more than any other work that lies within the range of his talents and opportunities. In the last analysis, it is his self-ideal that determines what work he will choose.

The fact that the vast majority of men (and women) do not choose their work in the full sense of the term but have to take what offers and are forced into jobs for which they have little liking and sometimes small aptitude does not change our interpretation. The opportunities available in a given culture may restrict a man's choice or make it difficult, but they do not make it impossible to choose. The most intelligent Hopi Indian cannot embark on a scientific career unless he breaks into the white man's culture and gives up his own, and even then the obstacles may be insuperable. A boy may be gifted like Michelangelo, but if he grows up among the very poor he may be forced to become a bricklayer or a ditchdigger and turn his hand to anything that offers. But the Hopi Indian and the gifted ditchdigger can still decide whether they want to abandon their bent or seek opportunities to follow it, no matter what the cost. In spite of all their efforts, they may not succeed. Or they may decide that the goal is not worth a life of hardship, sacrifice, and possible failure. But in any case, it is their decision whether to try or give up.

What limits man's self-ideal and curbs his ambition is not so much the lack of opportunities as the approval or disapproval of his group. This restraint begins at a very early age. A girl must not be interested in "boyish" games, from tree climbing to Meccano sets. A boy must not be a "sissy" and is taught by scorn and ridicule to channel his interests into sports and athletics rather than into art, nature study, or other intellectual pursuits. Murphy remarks:

> Watch children as they respond, for example, to nature study *before* they learn that it's sissified to be interested in birds and bugs. Or watch their interest in the world of creative activity, from making boats to making pictures, *until* they learn forms of social disapproval or the rules which define taboo on such activities, or a sense that these things belong to another kind of people. (1958, p. 182; Murphy's italics)

Not so long ago, a girl was supposed to be "beautiful but dumb" if she wanted to attract men. Consequently, a girl's self-ideal was more likely to be patterned after Mata Hari than Madame Curie.

There is a similar taboo, except for a few minority groups, on any kind of religious interest and practice. For this reason, religion has almost ceased to be a factor in shaping the self-ideal of a large portion of our people. In its stead has grown up another set of convictions, originally propounded by social scientists and propagated by writers and lecturers, to the effect that man is basically irrational, a creature of his appetites and emotions, and that he should not be censured for expressing them. If accepted, such a conviction cannot help but influence the individual's self-ideal and guide his actions. A man can bear the discomfort of unfulfilled desire as long as he believes there is a good reason for it. As soon as he sees restraint only as a social edict, a remnant of unenlightened bigotry, he rebels.

Thus a man's motivational system is established and organized around his self-ideal. As his ideal becomes integrated and articulated in his everyday activity, it determines his further actions. Whenever anything seems attractive or pleasant, it is also appraised as it contributes or detracts from the ideal toward which he aims. If he realizes that an action is bound to hinder his aspirations, he has to decide whether he wants to court a setback for the sake of some immediate pleasure or whether he wants to deny himself for the sake of his real aim. The prohibition against striving for something attractive that is not in line with his self-ideal is not an external mandate, nor is it a childhood taboo. Rather, it comes from his realization that these two goals are incompatible.

A man's self-ideal not only organizes his work or professional life, it also determines his development toward maturity. In fact, maturity means forming a valid self-ideal and living it. Whether farmer or merchant, teacher or artist, as a human being man strives for human perfection; and human perfection has its own stringent demands. What distinguishes man from the brute, and therefore must make for his distinctive human perfection, is his ability to understand and reason and his ability to act according to this understanding. Human perfection must be found in a self-ideal that is formed according to the best a man knows and understands and in actions that will actualize this ideal.

Self-expression as an ideal. Sometimes it is said that a man's potentialities are actualized by "self-expression." Discipline is scorned and the "sincerity" of emotional expression is exalted. But indulging one's appetites and emotions leads to surfeit and boredom, not to happiness or harmony. Expressing his emotions, pursuing each passing attraction, a man's "better self" remains unexpressed and unfulfilled and will revenge itself on him by an indefinable malaise, leading to cynicism and unhappiness. "Free love" may seem a worthy ideal, and sexual or emotional attraction may provide temporary satisfaction. But sex appeal is fleeting, charm of manner may fade. For a human being, love requires a personal relationship. What one person can mean to another cannot be grasped or exhausted in a casual contact. Sex without such human love is less than a second best. Thus our vaunted sexual freedom results in discontent and restlessness, as the many divorces for "incompatibility" attest. It is no accident that young people who permit themselves complete sexual freedom show appalling cynicism, boredom, and discontent (Arnold, 1950).

Self-expression as an ideal is even more dangerous during childhood when emotions are urgent and discipline is difficult. It is during growth and development that the self-ideal must be established and refined so that it can serve as a focus for the child's (and later the man's) actions.

The rationale of self-expression is the notion that a man should exercise all his functions. But in practice what is expressed is usually what is most urgently felt. Functions are exercised in doing and making things, in striving after some goal. Unless the goal is worth while and points beyond the child's horizon, there will be no challenge and no development. When the child's ideal becomes self-expression, he expresses mainly what he likes and dislikes. He scorns discipline and so fails to find the interest that would arouse his wholehearted allegiance. Such interests grow only with patient effort.

Young people even more than adults respond to an appeal that promises genuine achievement at the price of "blood, sweat, and tears." They admire hardihood, courage, even self-denial, for a worth-while cause. If all they know are Westerns and space operas to satisfy their love of high adventure, we cannot blame them if their self-ideal is formed after the pattern of the big-league football hero admired by all or the godlike space-man scorning the groundlings' conventions. At the same time, the ideals that could inspire young people must offer more than Horatio Alger did, or their goal in life will be one of physical prowess and sheer competitive success.

Unfortunately, such a self-ideal of naked self-expression and ruthless competition exacts payment in discontent and unhappiness. The child who is permitted a free outlet for all his impulses may be boisterous but is not a happy child. When he finds that adults do not resist his demands, his demands increase, but so does his unhappiness. The vandals in their early teens who make a teacher's life miserable and find their fun in destroying property are just as unhappy and perhaps more so than the timid subdued children of an earlier generation. Unless the right self-ideal is established, and established early in life, the youngster will be dimly aware that something is out of joint, that his conduct satisfies neither himself nor anyone else. At least in our culture, he cannot escape the knowledge that there is a standard of human conduct to which he ought to conform. If he refuses to do so, he retreats from the community of mankind into a ghetto of his own making. The approval of a gang or clique cannot blot out the knowledge that he should conform to a rule acknowledged by the company of truly admirable men of all time. The awareness of going against this rule may be successfully ignored but it leaves a malaise that cannot be appeased.

According to Lauretta Bender, unhindered self-expression is the mark of the child who has not had the continuous affectionate care of a mothering adult during the first year or two of his life, and hence has not been able to establish a self-ideal. Such a child remains uncontrolled:

> The child is driven by inner impulses which demand immediate satisfaction. . . . Motivation, discipline, punishment and insight therapy have little effect. . . . Screaming, kicking or temper tantrums or disturbed behavior of which the larger child is capable, continue when frustration occurs, as it must a good deal of the time. (1950, p. 123)

Apparently, these children do not feel guilt and show no anxiety, nor do they develop neurotic defense mechanisms. In psychoanalytic terms, they do not develop a superego. Yet if the experience of punishment were responsible for the punishing aspect of the superego, as psychoanalytic theory contends, there should be some one of all the punishing adults who could be selected as the "source of decisive frustration" and become incorporated as the superego. The fact that these children did not develop a superego is surely evidence that a positive self-ideal, formed because the child loves and admires someone, is necessary if he is to exercise self-control and develop normally. Without such a positive self-ideal, a child cannot measure himself and his behavior against someone else and the actions of that person which he knows are right. He continues to follow his emotions wherever they lead him and his personality remains unorganized and infantile.

10. THE SELF-IDEAL AND HUMAN CONDUCT

Though the self-ideal is chosen at first unwittingly and only much later corrected by reflection, it is not purely subjective. In so far as his ideal reflects the best that can be achieved by a particular individual, given his endowment, his assets and liabilities, it is objectively valid as the end of his development. Each person can only aim at (and achieve) *his* perfection, the perfection of both his individuality and his humanity. When a man's self-ideal is mistaken or perverted, he is in fact working against the deepest requirements of his own human nature. Whenever we use any of our functions inappropriately, we feel disharmony and malaise, whether we try to read in the dark or move our muscles in a way that brings pain. Similarly, we shall be neither happy nor content if we choose a self-ideal that is less than our human nature can achieve. Just as monotonous work brings boredom to someone who is too intelligent for it, so aiming for anything less than the best that is in us will soon bring ennui, malaise, disillusionment, and unhappiness.

CONSCIENCE AND THE SELF-IDEAL

A man may choose a self-ideal that is objectively valid and represents ideal humanity as it may be achieved by this particular individual; or he may choose a self-ideal to suit his own convenience, to satisfy a particular emotional bent. If his self-ideal is objectively valid, he will experience conflict whenever he is tempted to act contrary to his ideal. If he solves the conflict by acting as emotion prompts, he will be aware of guilt and suffer regret and remorse until he resolves once more to follow his ideal. If he tries to prevent remorse by changing his self-ideal, if he lowers his sights to accommodate a momentary attraction, the conflict will become unconscious and virulent.

The state of guilt. When a man has done something that is contrary to an objectively valid self-ideal, he is *guilty*. Such guilt is not an emotion, it is the state of a person who has broken a valid rule of conduct, whether he admits it or not. Rules of conduct may be merely social conventions (like introducing a younger to an older woman), they may be state or federal laws, or have the force of moral law.

Rules of conduct are not capricious; they are reasonable means for insuring good mutual relations among men. They are established either by informal agreement (e.g., polite manners), by formal edict (e.g., traffic rules), or by the inherent design of human nature (moral law). Since a valid self-ideal represents the hoped-for perfection of individual human nature, this self-ideal includes conformity to the moral rules that will insure right relations between men.

A rule of conduct is always obligatory, either for a certain group (saluting their superior officers is obligatory for all soldiers), or for the citizens of a given state (e.g., state laws), the citizens of several states (e.g., federal laws), for a whole culture (e.g., wearing black as a sign of mourning), or for humanity at large (e.g., not to hurt others maliciously, to care for one's children). Since rules of conduct are designed to guide men in their relationships with others, they cannot be abrogated at the whim of an individual, nor can they be safely ignored. When they are disregarded, the bond between the individual and his group is broken; in fact, the whole structure of the group is threatened, its smooth functioning is jeopardized. Consequently, every rule of conduct has penalties attached to a breach.

Legal penalties are designed to repair specific damage when possible and to restore the common bond. Thus the punishment is made to fit the crime: in primitive law, it was "an eye for an eye" and "a tooth for a tooth." Now once a law has been broken, an offense committed, it cannot be undone; nor is it possible to make the offender pay in kind and to the last farthing. For this reason, the lawgiver must determine the penalty which the group will consider a just retribution.

Legal penalties never exactly equal the offense, but the one offended in the person of the state declares them to be sufficient. In the nature of the case, the greater the offense, the more inadequate will be the punishment in strict justice. If the public authority nevertheless declares it to be sufficient and restores the offender to his position before the law as innocent (*non nocens legi*), it must mean that forgiveness is added to the penalty to square the account. The power of the executive authority to bestow juridical forgiveness is even clearer when a sentence is commuted or a pardon is given. When the transgression is serious, forgiveness is always necessary because full restitution is impossible.

Since guilt is incurred when a rule of conduct is broken, it is an objective state of affairs. But to be aware that he is guilty a man must know the rule and must acknowledge that it is generally valid. This is generally considered the function of conscience.

The judgment of conscience. The capacity to form a self-ideal implies that the child must be able to assess his own actions and decide whether they conform to this ideal. Such assessment is necessarily at first immediate and intuitive, comparable to the appraisal of any action as suitable or unsuitable. Gradually, however, the child also becomes able to assess his actions by reflection, on the basis of abstract principles. There is no doubt that his judgment is guided and trained first by his parents, later by everyone with whom the child comes into contact and from whom he learns what is considered right and what wrong. Because the child admires and loves his parents, he is willing to do what they think is right and so acquires a conscience as well as a self-ideal. Granted an inherent capacity for judging that some actions are right (not merely convenient, expedient, or commanded) and some wrong (not merely inconvenient, forbidden, or punished), this judgment of conscience must be provided with content, it must be trained. That some things are right and others wrong is admitted in every culture. But what is considered right and what wrong depends on the extent to which a cultural group, a family, or an individual is willing to consider the effect of various actions in their larger implications.

That we can make such a judgment of right and wrong is no more surprising than that we can judge whether something is true or false. Both are reflective judgments which complement the immediate intuitive appraisal. The judgment of conscience, deciding whether an action is right or wrong, complements the intuitive appraisal of this action as suitable or unsuitable, just as the judgment that a statement is true or false complements the intuitive sense judgment that it is long or short, that it sounds pleasant or unpleasant.

Since conscience is a judgment that compares one's own intentions with a generally valid rule, it depends on various factors, for instance, on whether a man knows that a law actually exists and to what extent it is binding. Only when a law is universally valid and binding can it be called a moral law. We usually speak of conscience only when we judge what is morally right or wrong. In an earlier publication (Arnold and Gasson, 1954) we have suggested how such universally valid rules of conduct can be derived from the demands of human nature. To give but two quotations:

> From the manifestations of the nature of an object in its actions, we can form some idea of what this nature demands. We know that human nature is different from animal nature—it is a rational nature. (p. 120)
>
> A rational nature demands of its very self that the rights of others be respected, that another's proper functioning is not interfered with, that one man help (and not hinder) another to achieve his final end. (p. 118)

Like any other judgment, the judgment of conscience may be mistaken. It may be based on insufficient evidence, or on an erroneous inference from sufficient evidence. There may be groups of people or whole nations whose judgment is wrong: for instance, the German National Socialists who considered it a crime to be a Jew; the Communists who made it a crime to be a kulak; and the primitive tribes who practiced cannibalism. It does not require much reflection to see that in all these cases the dignity of the human being is denied, he is literally treated as if he were an animal, absolutely subject to the will of his masters. In punishing Jews and kulaks, in killing and eating other human beings, man shows a disregard of human worth and freedom that is unworthy of human nature.

That the moral law is a demand of human nature needs little proof. Whenever it is disregarded or deliberately broken, human nature suffers and cannot reach its truly human perfection. *Bigamy* (or extramarital relations) make an intimate personal relationship between man and wife impossible. Here above all, "two is company, three is a crowd." It is significant that cultures in which plural marriage was practiced have lagged behind in recognizing women as individuals with all the rights and privileges of citizens. Once women are so recognized, plural marriage becomes obsolete. *Murder* destroys trust between man and man. A murderer cannot help but feel that every man's hand is raised against him and may find it necessary to kill again and again. *Breaking treaties* destroys trust between nations—which cannot be restored by diplomatic skill. *Cruelty, theft, and robbery,* which interfere with another's person or property, break the bond between men. The pleasure that may come from feeling superior to others is no substitute for the lost security of affection. *Self-indulgence* in food, drink, or sex leads to satiation and boredom.*

Since morality is founded in human nature, it is the basis of every other law. External law merely regulates the relations of citizens with each other. It judges and punishes actions, not intentions that do not lead to actions. The law of conscience, however, exercises no external coercion. Human nature demands not reluctant and rebellious conformity but wholehearted intention and action. Consequently, conscience judges not only actions, whether secret or public; it first and foremost judges intentions, even though they may never be carried out.

Men differ in the extent to which they know the demands of human nature and in the way in which they interpret them. They also differ in their willingness to act in accordance with these demands. We find men with a tender conscience who judge themselves culpable when they have broken the least of these rules and who suffer excruciating remorse; and then again men with a lax conscience who permit themselves actions that grossly transgress moral laws, but who have no awareness of guilt.

Guilt and guilt feelings. There may be objective guilt with or without guilt feelings, just as there may be guilt feelings with or without objective guilt. "Guilt feelings," so-called, merely indicate the subjective awareness of guilt, but this awareness may be based upon a mistaken judgment.

If a man erroneously judges himself guilty when he is not, he will have the same emotions he would have if his conscience were right. He may suffer from these emotions, but he will not suffer the consequences attached to breaking a law of his own nature. On the other hand, a man who judges himself blameless when he is in fact guilty will avoid unpleasant emotions but has to suffer the psychological consequences of his actions. Whatever he may think of cheating, in business or in marriage, even if he thinks it part of his legitimate self-expression, such dishonesty will in fact destroy the mutual trust between business associates or marriage partners. Even if the other does not find him out, his own actions cannot help but make him feel that others cheat as he does. With such an attitude, loyalty and trust are impossible.

In modern psychotherapy, guilt feelings are often thought to be completely subjective, the remnants of parental prohibitions internalized in the superego, or the irrational residue of once-valid taboos that are no longer binding. A patient is thought to have guilt feelings when he feels he has done something wrong or punishable, is afraid of punishment, yet seems unconsciously to want it—a thoroughly paradoxical state of affairs. In psychoanalytic theory, guilt feelings are thought to anticipate punishment by inflicting pain on the ego. Fenichel, for instance, explains:

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* This is a simplified statement of a very intricate problem. It must not be taken to mean that the problem of morality and culture has no wider ramifications than appear in this paragraph.

> Guilt feeling proper—that is, the feeling "I have done wrong," a painful judgment about some past occurrence which has the character of remorse—must be distinguished from feelings of conscience . . . [which have] a warning function. . . . The warning conscience says: "Avoid this or that action, otherwise you will experience a feeling of annihilation." Guilt feeling proper is more or less a materialization of this threat. . . . This feeling of annihilation must be characterized as a cessation of the narcissistic supplies which were initially derived from the affection of some external person and later from the superego. (1945, pp. 134-35)

In Fenichel's formulation, guilt feelings are "feelings of annihilation" which represent a loss of love, while "feelings of conscience" seem to be the fear of being punished by loss of love.

But feelings of guilt are more than anticipated punishment, more than fear of a loss of love from the superego. Even calling them "feelings of annihilation" does not convey their desperate urgency nor does it explain the so-called "need for punishment" that accompanies them. Guilt feelings are often the dominant symptoms in neurosis and are responsible for much of the neurotic's suffering. Guilt feelings often resist analysis, a fact first pointed out by Freud himself. For this reason, some psychiatrists, notably Trigant Burrow (1949) and G. B. Chisholm (1946), have considered guilt feelings the greatest scourge of mankind and have denounced the very notion of right and wrong because it is at the root of such feelings.

Chisholm, for instance, claims that morality discourages the use of patent facts on which to base our actions, prevents the rational use of intelligence, encourages men to believe something contrary to evidence, produces inferiority, guilt, and fear, and prevents them from sympathizing with others. He asks:

> Is there any force so potent and so pervasive that it can do all these things in all civilizations? There is—just one. The only lowest common denominator of all civilizations is morality, the concept of right and wrong, the poison long ago described and warned against as "the fruit of the tree of good and evil." (1946, p. 7)

In the "old Hebrew story," as Chisholm puts it, it was God who warned Adam against eating the forbidden fruit; and Satan (the "father of lies," as he is often called) who promised that it would make them like gods, knowing good and evil. Adam and Eve did gain a firsthand acquaintance with evil (which is the meaning of the biblical term "knowing evil"), but they were punished instead of becoming like gods. It is a rather curious twist to take the devil's mocking double-talk for sober truth and to assume that God's warning against sin was a warning against morality.

At any rate, neither in the Garden of Eden nor in modern life can the fact of a prohibition be the cause of guilt. It is not the law that makes criminals. The law is but a codification of the way in which human beings can live together amicably. In small groups, such ways of living together do not have to be set down and interpreted. In large groups, such as a civilized state, they do. Similarly, the moral law is but a set of rules for living as human beings. Morality is not a law imposed and enforced from outside by a wrathful God, as Chisholm implies; it is the conduct appropriate to our human nature. And that nature did not develop by accident. Even evolution presupposes that there is an impetus in nature that drives it not only forward but upward, from inanimate matter to man who can understand nature and work with it instead of being its witless tool. This creative evolutionary impulse can be blind as little as it can be accidental. It must have a source and an aim if it is to result in human beings who can come to understand nature and themselves. The moral law is found in human nature but it was not established by human nature. Man is not self-creative. The religious man assumes that moral law is established in human nature by the creator of that nature:

> Actually, divine law, as proclaimed in the decalogue, merely coordinates [man] with what his human nature requires. Divine law is merely a guide for man to understand his true nature, his true self and what is truly good for him. Hence divine law supports the effectiveness of the law of human nature. (Messner, 1950, p. 71; my translation *)

[*] Messner's original:

"Tatsächlich ordnet das göttliche Gesetz, wie es im Dekalog kundgetan wurde, nur den Menschen auf das hin, was die menschliche Natur selbst von ihm verlangt. Das göttliche Gesetz ist daher dem Menschen nur ein Führer im richtigen Verstehen seiner wahren Natur, seines wahren Selbst und des für ihn wahrhaft Guten. Demnach unterstützt das göttliche Gesetz die Wirksamkeit des Naturgesetzes im Menschen."

Similarly, Thomas Aquinas says: "Lex divina profertur homini in auxilium legis naturalis" (Summa contra gent. 3c 117).

Whether a man acknowledges the divine endorsement of morality or not, every man acknowledges as valid some rule of conduct that will insure mutual benefit. Even the criminal who breaks a law whenever it suits him, acknowledges some rule that holds between his associates and himself and feels guilty when he breaks it. The very fact that he distinguishes between those who can expect honest treatment from him and those who cannot betrays his dependence on a rule, however arbitrary the way he sets it up.

Unconscious conflict and the self-ideal. To abrogate a universally valid moral law for his own convenience means a change in a man's self-ideal that will produce an unconscious conflict which prevents contentment and happiness. An unconscious conflict in this sense essentially implies a clash between a man's potential natural development and his self-will. Here as in conscious conflict, there is a clash between two action tendencies, one the inarticulate urge to better himself, the other the explicit wish to find a short-cut to fulfillment and immediate satisfaction. The psychological consequences of changing the self-ideal are the same as those that follow upon transgression of the moral law: in both cases man acts contrary to his human nature and has to pay the price.

There are other types of unconscious conflict also. History tells us that man has always had desires that clash with the way of nature: he has always wanted security of life and limb; freedom from want; emotional security; intellectual adequacy. With all our advances in science and engineering, modern life offers no complete fulfillment. Civilization has secured the life and limb of peaceful citizens but has also prepared the H bomb. Marxism promises economic security for everybody in exchange for the most appalling insecurity in other areas of life. Psychotherapy offers emotional stability provided human demands are severely limited. Existentialism exhorts man to take courage even though he knows his helplessness in the face of nature. Logical positivism offers an answer to every question if only we agree that most questions are "meaningless."

The old Stoics offered a solution, no less radical, by urging man to school himself in denying his desires and cultivating indifference. Schopenhauer insisted that these desires are bound up with the will to live itself; this will must be denied and negated before the desire for the impossible will cease to torture us. From the many remedies offered, it seems that the desire for possession, and permanence in possession, for enduring love and perfect knowledge, is part and parcel of our human make-up, bound to lead to permanent frustration if it can never find fulfillment.

For these frustrations, science has no solution. They form the background to everything we feel and do; yet they remain unconscious and rarely attract attention. They result in a pervading attitude of uncertainty and diffidence: the existential anxiety we have discussed in Chapter 12 of the preceding volume. The religious man claims to have an answer to such questions. He would say that the very fact of such universal desire for unfailing love and understanding, for enduring happiness, would indicate that it is to be found somewhere, if not in this life then in the next. Though men cannot satisfy a need they have not created, God the Creator can and will. The partial and temporary security found in this world is but the promise of an enduring and full satisfaction. The happiness of friendship, the delight of the good things of life are but a token of the happiness of loving God who is all in all. Recognizing the token character of the joy in human companionship and the pleasures of good health and physical comfort, even of artistic, scientific, and cultural satisfaction, man can endure their impermanence without frustration and suffer their lack or loss without despair. The Christian would add that even the choice of a valid self-ideal is difficult without faith in God. The most admirable man has glaring imperfections that detract from his value as a model; and the highest ideals are not attractive enough to justify the endless discipline necessary to act consistently as the ideals demand. Only when God is recognized as the ultimate goal and His only Son as perfection incarnate, does it become easy to establish the proper hierarchy of values and to follow this model of human perfection.

Whatever we may think of this answer, it does offer a solution to man's unconscious conflicts and anxieties. With faith in God, the self-ideal finds its natural end and support. With faith in God, there can be no existential anxiety, no pervading fear of want, and no enduring sorrow.

Whatever our solution, we must organize our personality according to a valid self-ideal if we are to escape the consequences of emotional indulgence. To go against the demands of our human nature by indulging our emotions instead of letting them support our human purposes, is to bring upon ourselves a pervading discontent and unspoken distress. To strive toward a self-ideal that does not perfect human nature is not worth the effort. To suppose that human nature can stand permanent frustration of its deepest desires is to court ultimate despair.

EMOTIONS, THE GUARDIANS OF THE SELF-IDEAL

A man's progress toward his self-ideal, revealed in his deliberate choice of action, may suffer many a setback and need many a fresh start. He is tempted by pleasure, by gain, even by love or friendship to do what he knows in his heart is not what he ought to do. When he has done something he judges wrong, he will experience a variety of emotions that urge him to repair his action. Conversely, his progress toward his self-ideal is supported by positive emotions, by his love for everything that is good, true, and beautiful.

Embarrassment and shame. When a man realizes that he has somehow fallen short of what he believes himself capable and thus has lowered himself in his own esteem and that of his fellows, he feels embarrassment and shame. Buytendijk (1950) suggested that shame is felt when a person is discovered, unmasked, and his unworthiness exposed. And Nuttin (1950) pointed out that the human being needs and wants privacy and feels shame when this privacy is violated. Similarly, Helen Merrell Lynd (1958) noted in a tender and perceptive analysis that shame is the exposure of oneself and one's society, and must be faced if a person is to live beyond the conventions of his particular culture.

A man wants to communicate with others, but he does not want to communicate every thought and notion. Everyone wants to be secure in the affection and esteem of his fellows, and an exposure of himself as he knows himself in the privacy of his own thoughts cannot but be precarious. We are both better and worse than others know, and are embarrassed not only when we appear in an unfavorable light but also when our tenderest impulses are exposed to public view.

Both embarrassment and shame occur when a man believes himself exposed to an unfavorable judgment. But *embarrassment* is primarily a reaction to being found out by others, whether in an act of virtue or of vice. Thus a man may be embarrassed because he has forgotten to take off his hat on entering a church, or because he has been found gullible, but also because he has been caught praying or giving money to a beggar. Embarrassment seems to follow his estimate that he has lost face with the person or group to which he belongs. If this group is intellectual or arty, he will be embarrassed if he has inadvertently betrayed an interest in baseball or wrestling. If his group is sporty or tough, he will be embarrassed if he is caught reading poetry. Everyone has his circle of friends and acquaintances and is aware of their scale of values. To win and hold their esteem, his conduct must conform to their ideal, even though he is well aware that his inner life, at least in some respects, may not.

The term *shame* is often used in the same sense as embarrassment, as when we say that somebody is ashamed of being poor, or ashamed of a physical defect, or ashamed of not knowing the right pronunciation of a word. A girl may be ashamed of wearing clothes that are not in style. Here, as in embarrassment, there is an exposure of oneself which reveals that the self does not come up to the social ideal of being comfortably off, healthy, and well educated. In embarrassment proper, there is perhaps an added element of surprise: it is the sudden exposure to criticism that brings a blush. This element of surprise or startle may account for the parasympathetic excitation which results in a sudden rush of blood to the face (see Ch. 7). Embarrassment is shown not only when we inadvertently expose ourselves to ridicule or blame, but also when others invade our privacy by insisting on such exposure.

That such embarrassment has nothing to do with right or wrong conduct is shown very convincingly by some clinical observations. Rollo May (1950), for instance, reports that there are patients who show neither resistance nor disturbance when talking about sexual, aggressive, and egotistic impulses, but who are covered with confusion when they have to admit tender or compassionate feelings. Of the same order is the embarrassment so often seen among people today when God or religion is mentioned or when it is suggested that the human being can act from moral aspirations or a love of God. Such an attitude makes it clear that the self-ideal commonly acknowledged is that of the red-blooded, "hardheaded" self-made man who lives out his impulses aggressively, unfettered by moral or religious scruples. He may indulge in romantic infatuation but is proudly autonomous and has no need of God.

We speak of *shame* in the narrower sense when we are revealed as blameworthy or culpable (not merely as ridiculous, foolish, or undignified). This need not mean being *caught* in a culpable act; it may also mean that we ourselves are realizing our culpable tendencies. A man can be ashamed when he is caught stealing, but he may also be too ashamed to steal. It is the latter kind of shame that aids in striving toward a valid self-ideal. Once the self-ideal is formed, once excellence is known and wanted, shame prevents a man from acting contrary to this ideal. Shame in this sense is aroused whenever the exposure of self reveals it as inferior to the ideal or whenever the human being is revealed as body rather than person. From this point of view, it becomes understandable why nudity arouses shame among all civilized peoples. In fact, even native tribes in tropical climes usually have a minimum of covering. The nude human body gives the impression that the body is dominant and the person submerged; clothing prevents distraction from the face which expresses the person. We are told that Adam and Eve were ashamed of their nudity after they had sinned—a subtle reminder that sin had deprived them of their former control over their instinctive impulses. From now on, it was as difficult for them as it is for us to act rationally, against strong emotional attraction; nudity now reminded them, as it does us, of the insistence of bodily appetites, of the difficulty to remain truly in command when the body draws too much attention. Scheler puts it this way: "Only because the body belongs to man essentially can he come to the point where he *has to be* ashamed; and only because he experiences himself as a person essentially independent of the body . . . can he come to the point where he is *able* to feel shame" (1913, p. 69; my translation[*]).

Both shame and embarrassment are aroused by an appraisal that the person is not acting in accord with human ideals. Shame is primarily the reaction to loss of *self-esteem,* for it indicates that the self does not measure up to the self-ideal as it ought to be, while embarrassment is mainly the reaction to threatened loss of *esteem from others.* Others may esteem us for acting according to an objectively valid self-ideal if it also represents the group ideal; but the group ideal may be severely limited and may even contradict some of the demands of human nature (e.g., the ideal of the swaggering "toughie" popular in juvenile gangs). It could be said that there is a type of shame that is rooted in a man's essential human nature, while embarrassment is always rooted in his relationship to his group. No matter what the moral climate may be in any culture, shame is a universal phenomenon and a strictly shameless person is thought inhuman in any age.

[*] Scheler's original:

"Nur weil zum Wesen des Menschen ein Leib gehört, kann er in die Lage kommen, sich schämen zu *müssen;* und nur weil er sein geistiges Personsein als wesensunabhängig von einem solchen 'Leibe' erlebt, . . . ist es möglich, dass er in die Lage kommt, sich schämen zu *können.*"

Of course, neither shame nor embarrassment are simple emotions. They have elements of sadness and dejection combined with a fear of loss of love or esteem, as well as a fear of the consequences of one's action. There is no reason why animal pets who are devoted to their master should not feel sadness and fear of losing the master's love. Surely, scolding a dog is so effective because the dog is devoted to his master. Scolding a young puppy or a strange dog has no effect unless it is accompanied by punishment. The dog who has stolen a piece of meat gives every sign of being ashamed, although it would perhaps be more accurate to say that he is embarrassed. He does not react to a loss of self-respect but to a loss of his master's trust and love.

There is no undue "anthropomorphism" in ascribing embarrassment to a dog. When the dog steals a piece of meat and makes away with it furtively, his furtiveness cannot be explained unless we assume that he knows he is doing something he is not supposed to do. When his master finds the meat gone and scolds him for it, the dog cringes, hangs his head, and slinks away. If he did not know his master was angry, he could never learn to leave the meat alone. And if he does recognize the master's reproach, he must know that he did something the master did not want him to do and now is sorry. The dog's reaction is very similar to the reaction of a small child who is caught stealing jam. Both child and dog are sorry for what they have done, not only because they expect punishment but primarily because they have been caught doing something they knew they were not supposed to do, and because it was parent or master who caught them. The difference between this reaction and the shame an adult or an older child experiences is that the latter can judge in addition that what he did was wrong (not only forbidden by someone in authority) and can feel shame even if nobody finds out.

Remorse and repentance. Of all the emotions that can be aroused as a result of a man's judgment that he is morally guilty, the most characteristic are remorse and repentance. There is shame, to be sure, for there is a conviction of having done something that is blameworthy. There is also a fear of being discovered, a fear of loss of esteem, and of possible punishment.

But in addition, the awareness of moral guilt arouses regret and remorse for having done something that is wrong and has harmed others. Remorse urges the person to rid himself of his guilt. He may merely wish he had never done what he knows is wrong—which is an unprofitable reaction.

He may blame himself, even rage against himself, but what is past cannot be undone. Or he may determine to repair his fault and make amends. But often there is no possibility of repairing the damage; then remorse will become intense, he will suffer the anguish of the "again bite of inwit," the "Gewissensbiss," which insistently urges that something be done.

Such remorse is felt whether there is true moral guilt or not, as long as there is a subjective judgment of culpability. Gasson recently reported an incident that vividly illustrates remorse as a reaction to such a feeling of guilt:

> Not so long ago a young gynecologist came to me to discuss an unfortunate incident that has happened to him in the very recent past. One of his patients, a woman, referred to him by another gynecologist, asked for his help because of her sterility during the twelve years of her marriage. In the course of this time everything that any gynecologist could think of to relieve her condition had been done and the young doctor had very little hope that he could help her . . . In the physical examination he was dubious about what palpation had indicated and asked the patient to come back in a week. On the second examination he suspected a fibroid tumor in the uterus and watched it very closely. It seemed to grow rapidly. To confirm his diagnosis, he sent his patient to another man. The other doctor agreed with his findings and recommended surgery. The patient underwent a complete hysterectomy because there was evidence of fibrosis in the ovaries. When the pathologist examined the uterus after excision, he found not only fibroid tumors but also a foetus. The young gynecologist was all broken up about this development. He felt extremely guilty for not having done a Friedmann [pregnancy test] before surgery. Now he felt guilty about something for which he was not morally accountable, though it is true that it was his advice that resulted in destroying the child the couple wanted so intensely, and further destroying any possibility of conceiving another. (Gasson, 1960)

Gasson comments that it is the desire to make reparation which cannot be fulfilled that accounts for the intense remorse of the young doctor.

Even where the harm can be repaired, it can never be undone. Punishment, as we have seen, does not square the account fully, either, because it can never be made exactly to fit the crime. To restore his old standing in the community of his fellows, a man needs forgiveness in addition to punishment and restitution. Forgiveness can only come from the one who is offended. When a child hurts his mother by a thoughtless word and realizes it, he can ask for forgiveness. He knows he deserves punishment, and he may even want it; but punishment is equal to the offense only if the one offended accepts it as such. It is only the mother who can either punish and declare herself satisfied, or who can forgive even without punishment. Without her forgiveness, the child remains guilty and knows it. He may react to the guilt by asking for forgiveness belatedly, by bravado which forces punishment, or by doing nothing and carrying with him the conviction that he is guilty, deserves punishment, and is bound to experience it some time, somewhere. But short of being forgiven, the mutual harmony is broken.

Moral guilt is the result of an offense not only against others who may have been wronged, but also and always against a man's own human nature and its creator. Unless he seeks forgiveness where full forgiveness can be found, his guilt cannot be expiated and he will continue to seek punishment, whether consciously or unconsciously. For this reason, neurotic feelings of guilt cannot be removed effectively until the patient acknowledges real guilt. When this is expiated and forgiven, the common bond is restored and he can again feel at ease and at peace. To reduce morality to man's autonomous opinion takes away the neurotic's last hope of being punished for his guilt and so of setting himself right again. His desperate play for retaliation is his last effort to appease his conscience.

Once the patient's real guilt is expiated, his "guilt feelings" can be reduced by therapy. Remorse, like fear or any other emotion, may grow into an enduring attitude, either as the result of a traumatic experience (such as recounted by Gasson) or as the result of many occasions on which a man has been aware of guilt though he has never been found out, has never made restitution, has never experienced forgiveness. Since conscience judges intentions as well as actions, this sense of guilt may occur even though the offense has merely been rebellious resentment and concealed ill-will. The guilt feelings neurotics often experience toward their parents seem to be the result of remorse for their ill intentions as much as for their past actions. They know themselves guilty because the command to "honor your parents" obliges even though parents may give cause for resentment. Neurotic "guilt feelings" are rarely the result of justifiable rebellion against unreasonable parental demands but rather the result of bitter resentment aroused by the parents' often necessary interference with their children's wishes. As fear may grow into an attitude of anxiety, so remorse may develop into guilt feelings that are diffuse and "free floating," without recognizable connection with the roots from which they spring.

Unlike anxiety, they cannot be corrected by convincing the patient that what was guilt when he was a child is guilt now no longer. They can be eradicated only when the core of real guilt has been isolated and expiated.

Cameron (1951) has suggested that the patient who is unable to express anger and assert himself and who "suffers from covert resentment about which he feels guilty" may be helped by training him to express his anger openly and unrestrainedly, particularly against the "key person" who is responsible for his passivity. But if Cameron is right when he observes that such patients feel more and more guilt the more their covert resentment increases, it is unlikely that they will not feel guilt when they give unrestrained expression to this resentment. If the ill intention of covert resentment is culpable, so is the open expression of this resentment, when the same moral standards are applied. What seems to happen in Cameron's procedure is that the patient's judgment is changed so that he now considers right what before he considered wrong. This is confirmed by Cameron's statement that in the course of such training for free expression of anger he deals with the patient's "resistances and rationalizations": thus the patient might say his mother is old and not well and he does not want to hurt her, that everybody would blame him if he were to be angry with her, and the like. There is, of course, the possibility that unexpressed resentment might be more virulent and contain far more ill-will. In freely expressing his anger, the patient may restrain himself sufficiently to remain within reasonable limits while relieving the pressure of his anger. But that is unlikely because Cameron aims to have the patient relive the major occasions arousing his resentment so that the anger is reexperienced and ventilated. It seems a doubtful procedure to reduce the patient's guilt feelings by changing his moral values, which is essentially what Cameron's procedure seems to do.

Repentance or contrition includes not only regret for a wrong done but also the decision to do it no more. Without repentance, sorrow for wrongdoing is little more than a gesture. Even when this sorrow is accompanied by a fear of punishment, it is not enough to provide a motive for turning toward what is morally good. Such a positive turning away from evil and toward good is implied by repentance. It is a rational action tendency often supported by emotion. For the religious man, the desire for the good is an action tendency that is both rational and emotional. For him, the love of God has become personal, and repentance is a return to the Beloved. When forgiveness is asked and given, harmony between God and man, between man and his fellows, and between man and himself is restored and with it man's effective striving for his true self-ideal.

Self-blame or self-condemnation. When a man does not come up to his own standards, whether of morality or intelligence or competence of any sort, he is likely to blame himself for it, to be angry with himself. If he is inadequate because the demands made on him are too exacting, he may feel chagrin or despair, depending on his appraisal of how important this particular challenge is for him.

Rosenzweig (1934) noticed that some individuals habitually blame themselves rather than others when they are exposed to frustration. Others habitually blame the experimenter or the frustrating situation. Funkenstein and associates later found that "anger-in," as Rosenzweig called such self-blame, results in a physiological picture (as measured by ballistocardiograph, pulse rate, blood pressure, skin resistance, and similar indicators) which is closer to fear than to anger (see Ch. 7). This is understandable enough, for a man's appraisal that he is inadequate always arouses fear in addition to self-blame. Only when he can assume that the experimenter is at fault or some other external factor that could be overcome will he feel anger rather than fear. "Anger-out" expresses his conviction that it is not he but the other who is at fault. "Anger-in" is the result of an appraisal that he is inadequate, which induces fear, and secondarily provokes an impotent anger against himself.

Boredom. When a man is neither striving toward a goal nor enjoying its achievement, when he is neither doing nor making anything, when there is not even anything to love or hate, to fight against or to flee from, a peculiarly painful feeling takes hold of him: ennui or boredom. It is an emotion and urges to action, but it is the reaction to the *absence* of something that can engage his interest. Boredom is as "objectless" as the malaise that comes from unconscious conflict. Like such conflict, it is the result of a way of living that cannot actualize human potentialities. It is the reaction to a frustration of the human desire for happiness.

All human functions need exercise, not only sensory functions which, at first glance, seem to bring the greatest return for the effort. Not all functions can be exercised equally and not all goals toward which they lead are equally valuable. Being human, man must use his properly human functions if he wants human happiness: boredom is inevitable if he concentrates merely on pleasure and comfort. Without the joy that comes from doing and making, from having a creative purpose, from working with others toward a common goal, he is less and less satisfied with having more and more. Pleasures of sight, touch, or smell soon pall, food ceases to tickle the palate, sex without love becomes an empty pantomime.

Boredom indicates either that a man has not chosen the right self-ideal or that he is disinclined to make the effort to strive for it. The only remedy is constructive effort, which will create enthusiasm for a worth-while goal. Clearly, such effort will only be made if he becomes convinced that his way of life is unsatisfactory, or his self-ideal inadequate. Boredom is the emotion that makes his present way of life unbearable, and is the spur that may urge him to change it.

Human suffering. Our discussion of the role of negative emotions in striving for the self-ideal would be seriously deficient if we did not touch on human suffering.

We have seen that physical pain is a reaction to excessive stimulation (Vol. I, Ch. 4; Vol. II, Ch. 2). Both man and animal feel pain but a man suffers more because he knows that pain means disturbed functioning and he is afraid of the disease as well as the pain.

Excruciating though physical pain can be, the peculiarly human form of suffering is not physical. We suffer when someone we love is ill, struck by misfortune, in pain, or dying. We suffer when we lose someone we love, when we lose our health, our livelihood, our prospects of achievement or success. We suffer when we are unjustly accused or punished, when we lose the love or esteem of those we hold dear. We also suffer when we have offended someone we love, when we fight against doing what we know is wrong and yet do it. In short, we suffer when we cannot or do not lead a life that insures human happiness.

Suffering and pain may lead one man to despair and another to heights he could never have reached in any other way. Suffering is the great enigma, the challenge and test of man's fortitude. Down the ages, men have cried out with Job: "All that I was is gone, the ambition, the happiness that was mine swept away like clouds before the storm; my heart is dead within me, a prey to long despairs" (Job 30.15–17; Knox translation). And like Job, men have been disturbed by the suffering of the innocent and the prosperity of the wicked: "How is it that godless men live on, meet with advancement, enjoy their riches undisturbed?" (Job 21.7–8). "Tell me, how often in very deed are the hopes of the wicked extinguished, engulfed by the flood?" (Job 21.17).

Bearing suffering manfully may be the highest achievement possible to man, as Frankl (1959) insists. But it is doubtful whether suffering can bear positive fruit unless a man have Job's trust in God: "I acknowledge it, thou canst do all thou wilt, and no thought is too difficult for thee. I have spoken as fools speak, of things far beyond my ken . . . I have heard thy voice now; nay more, I have had sight of thee; now I am all remorse, I do penance in dust and ashes" (Job 42.1–6).

There is little doubt that so embracing his fate will give a steadiness to a man's life that nothing can upset. Suffering has challenged his values and found them true gold. Until it is tested through suffering, a man's self-ideal may be the casual outcome of an easy and successful life. Suffering seems to be the fire in which his self-ideal, and thus he himself, may be either purified or destroyed.

CONCLUSION

All the emotions we have discussed can help a man in his progress toward his self-ideal. Some are felt when he falls short of what he expects of himself (e.g., shame, self-blame); others when his hopes for the good things of life are frustrated (e.g., suffering); still others when he is disinclined to pursue a valid self-ideal (e.g., boredom). All these emotions urge him to change his way of life, but he need not change it constructively. Shame or remorse may lead to despair; great suffering may spawn rebellion and finally apathy; boredom may be allayed, at least for a time, by a round of distractions. For good or for ill, emotions influence a man's actions but cannot force them. To draw positive benefit from his emotions, a man must recognize their origin and decide to solve his problem rather than evade it. Shame or remorse or boredom will lead to a change for the better if he will resolutely strive toward a valid self-ideal; suffering can be borne constructively if he can see it as the unique chance to come to terms with his life, his death, and his ultimate end.

II. SOME POSITIVE HUMAN EMOTIONS

It is idle to speculate what an animal's experience of emotion may be like. Yet it is fatuous to deny that man and animal experience emotions that are in some way analogous. We have seen that we can recognize emotions in animals as well as in human beings, that we can predict their behavior during emotion, and that they respond to our emotions. Thus, we must be able to recognize emotion in animals and they must be able to recognize our emotion; this implies that such recognition is based on an immediate intuitive sense judgment.

Animals as well as men seem to feel likes and dislikes, to experience fear, sadness, anger; they seem to look forward to reward or expect punishment. But since emotions follow upon appraisal, they cannot be identical in men and animals. Even in their immediate intuitive appraisal, human beings can estimate aspects of things and persons that are not sensory qualities. For a man, sexual attraction is heightened by beauty of face and form, appetite is tempted by well-prepared and beautifully served food. Even the most basic emotions are intensified and sometimes aroused by factors that can only be appreciated by human beings. For this reason, the experience of emotion is bound to be different in men, though its core, the fact that some attraction or repulsion is experienced, may remain the same.

There are some things that can be appraised only by a reflective judgment carrying intuitive judgment along with it. Such appraisals arouse truly human emotions. Their objects have neither interest nor attraction for animals and the actions to which they urge are outside the animal's range. A man's enthusiasm for football or hockey, for instance, is based on an appreciation of the game and the way it is played, which is not an intuitive judgment. No animal could become a football fan. But given the knowledge of the game, and the judgment that it is important to win and important to play well, a man will experience emotion when a quarterback breaks through the line and gets away, or the goalie makes a brilliant glove save.

The fact that human emotions are aroused by objects or by aspects of objects which can only be appreciated through a reflective, i.e., a value, judgment does not mean that every value judgment will result in emotion. Emotion follows only upon a judgment that *"this particular thing is good or bad for me here and now,"* whether this judgment is intuitive or reflective. Value judgments that refer to a class of things (e.g., "criminals are bad men") or to something that has no relation to me or mine (threat of famine in Tibet) or has no immediate significance (the effect of fall-out two hundred years from now) will arouse no emotion. Of course, it is a quality of human experience that imagination can be directed at will to picture the situations so judged. The better informed the person and the more vivid his imagination, the greater is the possibility that even in these situations he will find something that concerns him personally: someone he knows may be a criminal; famine in Tibet may mean misery to men like him; atomic fall-out may be a dire threat to his children's children.

We have pointed out before that it is not possible to make an exhaustive list of human emotions, nor would it be very profitable. Since the particular value judgments that result in emotion can be so varied, there will also be great variety in the emotions experienced. However, it is possible to discuss some of the more frequently felt human emotions, always with the proviso that we are speaking of an emotional pattern with many individual variations, depending on the aspects that are appraised and on the action they seem to require.

There is little experimental evidence available for most of these emotions. In recent years, there has been a tendency to investigate theoretical concepts like transference, identification, projection, or empathy. While these possess concealed emotional aspects, their relationship to emotion is obscure and does not allow valid inferences. It seems curious that an empirical science should design its experiments for investigating theoretical preconceptions rather than examining and explaining experience. Perhaps the dissatisfaction with the results of such studies, voiced by acute critics like Cronbach (1958), will have a salutary effect and lead psychologists to formulate theories that show more respect for experience and less admiration for mechanical analogies. At present, our discussion of positive human emotions has little to gain from experimental studies but may profitably turn to philosophers for a phenomenological analysis which may shed light on what is one of the most important aspects of human life.

In the last chapter, we saw that some negative emotions can be used to shape life creatively toward a valid self-ideal. In a similar way, various positive emotions can make it easy to follow that self-ideal. These emotions, like basic emotions, may develop into attitudes; and these firm emotional dispositions give rise to the habits we call virtues.

THE DESIRE TO KNOW

We have seen earlier that the desire to know is the first action impulse aroused by anything new that is sensed and appraised (see Vol. I, Ch. 10, Interests). In this form, it has been called "spontaneous attention," "curiosity," "exploratory drive," etc. It is experienced by man and animal alike and leads to action: to looking, exploring, and manipulating. But human beings can experience a desire to know that goes far beyond elementary "spontaneous" attention. This desire to know will lead first to "voluntary" attention and next to systematic investigation: to reading, collecting data, experimenting, making informed guesses, reasoning, and other such activities, all of them possible only to beings who can form concepts and use symbols. "Interest" is the sentiment that develops from the desire to know, and leads to a preoccupation with a problem that continues for some time. When this preoccupation becomes systematic and leads to the well-integrated knowledge of a particular field, it becomes a habit of action with its own method of investigation. *Scientific method* is such a habit of action which finally results in a body of knowledge, a *science*.

When this interest is turned toward the general problem of human life, its conditions, its purpose, and toward facts as they affect the human being, it leads to *wisdom*, a virtue that is grievously underestimated in our age. The wisdom of the sage, more than the scientist's knowledge, leads to a realization of what is worth while in life, to value judgments that are informed, reasoned, and free from emotional bias. Such value judgments will bring an appreciation of human potentialities and will lead to a better knowledge of the self-ideal valid for human beings in general and for each individual in particular. Traditional philosophy, for good reason, has called science and wisdom *intellectual virtues*.

UNION WITH OTHERS

There is one class of emotions that is absolutely indispensable to the formation of the self-ideal and its successful pursuit. These emotions are the variations of human love. An animal can feel an attraction to things and living beings; but man can reflect on the meaning of the world around him and on the personal significance of others like him, and so finds his love illuminated by knowledge. His love and understanding unite him with the living beings that people his world and with that world itself in a way no animal can imitate.

Scheler (1923) has discussed various forms of love extensively. According to him, man feels at one with all life and thus experiences a real emotional identification. He finds such experiences of oneness in primitive thought, in various pathological cases, in hypnosis, but also in the identification of follower with leader, in the union of lover with the beloved, or of mother with child. It is Scheler's conviction that such emotional identification occurs below the level of waking consciousness, is involuntary and confined to the sphere of vital consciousness. On the ground of this "Einsfühlung," can grow the vicarious feeling ("Nachfühlung") which, to him, is necessary for genuine fellow feeling. Finally, such fellow feeling, a benevolence for everything human, is the basis of a genuine love for another person. In Scheler's view, the felt oneness with everything that is, expresses man's relatedness to nature and is fundamental to all his relationships.

This experience of oneness, the joy of communion with all living beings, certainly is emotional in character. But does it require a special process, "emotional identification"? If there is never a "cold" perception but always a perception followed by an intuitive appraisal that arouses emotion and leads a man to what is good for him, to everything that can enhance and perfect him, his love necessarily goes out to the world around him. The very fact that man depends on his world for all his needs and that he recognizes it as good for him, the very fact that he wants things, achieves them and enjoys them, that he makes things to use and cherish, implies an intimate relationship to his world. The very fact that he can flee from danger to *safety* emphasizes his belongingness, is proof that he is at home in the world. Unless he is badly frightened, starved, or deprived, he cannot help but love the world around him. Since this outgoing love is based on an immediate intuitive appraisal, it is "below the level of waking consciousness," as Scheler says; it is involuntary and confined to the sphere of vital consciousness. It can be frustrated by traumatic experiences and even by a rational appraisal that is all too concerned with the world as it is for scientific understanding, rather than with the world of nature as it affects us. The notion that the world has "physiognomic character" for the child or the primitive (Werner, 1940) merely means that sunshine, waterfall, landscape can be appraised simply, intuitively, as they affect the human being, just as animal actions or human expressions are appraised. Scheler rightly says that such an approach is not anthropomorphic but a genuine insight into nature, inaccessible to the man who uses only an inferential rational judgment.

With such a loving approach to the world, there is a sense of kinship, of belonging. Hence Scheler's "Einsfühlung" is the result of the basic intuitive appraisal that the world is good and offers us what we need. And since this is love, albeit a love that is expressed only in the confident bearing of men in their familiar surroundings, it necessarily is included in every other love. A man who did not have it would be incapable of any other love simply because even the most personal human love needs the immediate intuitive appraisal that is found here almost unmixed. But this most basic love is as yet no more than the natural attraction to everything that is good for him, an attraction shared by animals as well as men.

The other examples of emotional identification in Scheler's discussion seem to result from an imaginative absorption in another, rather than from emotion. This is true for various pathological cases mentioned by Freud, as for instance, that of a girl who developed the same cough as her mother, or that of girls in a boarding school who had hysterical fits when one of their group flew into hysterics on being jilted by her lover. A similar imaginative absorption is illustrated by the play of a small child who insists on being a dog, runs around on all fours, refuses to speak and barks instead, wants to eat from a dish on the floor, and the like. This is neither imitation nor emotion but rather an imaginative living as the other does, even if the other is a dog.

There is emotion in the ecstasy of loving sexual union, or of the mother nursing her child, but an emotion that depends on an immediate intuitive appraisal without interference from reflection. When union is complete, all desire is stilled and the lover rests in the beloved. There is a feeling of oneness because it is a moment when love is undivided and cannot be distracted, when all is joy. Yet this joy is so profound only because there is a personal love in which the other is appreciated not only as a partner but first and foremost as a human person.

The identification of a group with its leader is based on emotion also. In this case, it is admiration for the leader and the ideals he represents. While the admiration that moves the child to model himself after the father is aroused by an immediate intuitive appraisal, at least early in childhood, the admiration that leads to unquestioning loyalty to a leader is based on a reflective as well as on an intuitive estimate. Whatever the reason for the leader's appeal, it can be appreciated only by thinking about him and reflecting on his aims. The group may be carried away by the enthusiasm of its leader, but the leader must be on fire for something that seems appealing or worth while. Even the delinquent gang does not follow its leader for the sheer appeal of getting cars, food, or money. What really appeals is the power their exploits imply, power that is wielded by their leader and through him, by *them*. The emotion kindled by a leader may impel unquestioning, even fanatic, commitment and form a close bond between leader and follower. But this bond is forged by emotion and not by a separate process of "identification."

Infectious emotion. According to Scheler, both infectious emotion (emotional contagion) and empathy are special cases of emotional identification. But to explain why we become infected by a friend's depression or the gaiety of good companions we need not postulate a special process. There is no such concentration on another as would bring about a spontaneous similarity of expression, nor is there a deliberate decision to make an imaginative effort. Rather, the mood of gay companions will be expressed in gay conversation, and gradually the newcomer will pay attention to their way of looking at things until he eventually responds to their hilarity. Their high spirits will cheer him up, capture his attention until he begins to share their fun. "Emotional infection" is no more than a gradually shared emotion; the emotion is shared just as soon as the newcomer can appraise the situation in the same way as the others do.

The emotional object the newcomer gradually comes to share, together with his response to the others' emotional expression, accounts for all such emotional infection mentioned by Scheler. It can also account for the emotion that grips a lynch mob or a crowd during Mardi Gras. We respond to others not only as the persons they are but also as they are gay or sad, angry or fearful. Given the situation that has aroused strong emotions in them, it will do so with every newcomer. The appraisal by each member of the group will be reinforced by the mutual accord of which their emotion convinces him. Whatever restrictions each individual has accepted for himself, if he sees others act without restraint, sees them give in to their emotion, he may easily decide that it is safe to do so himself and act accordingly.

Empathy. A special case of emotional identification seems to be empathy. Lipps (1903) advanced the theory that *empathy* is the source of our knowledge of others, comparable to *sensory perception* which is the means by which we know inanimate objects, and *introspection* by means of which we know ourselves. Unwittingly, one person imitates the behavior of the other. This results in kinesthetic sensations from muscles and joints which in turn give rise to an awareness of the other's feelings. Since this imitation is unwitting, it gives the impression of a direct intuitive knowledge of the other. According to Lipps:

> Empathy is the condition of joy in the inner state of another as observed in his expression. . . . I am joyful with him whose joy I see in his expressive movements, I experience it immediately in observing his expression, unless such joy contradicts my own nature. I do this the more surely the more freely I contemplate his expressive movements, undistracted by irrelevant thoughts and interests. Also, I enjoy observing the anger or pain of another when I can participate in it freely, i.e. when my own inner nature is freely expressed in such anger or pain. (1903, I, 111; my translation *)

When the feeling expressed in the other's demeanor "contradicts my own nature," there is a strong sense of conflict, according to Lipps, from which one directly knows the feeling state of the other. The clearest case of empathy in Lipps's sense is that of the onlooker at athletic contests or acrobatic exhibitions. Lipps points out that the onlooker often duplicates the acrobat's movements, thus expressing his empathy.

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* Lipps's original:

"Einfühlung ist Bedingung der Freude an dem in der wahrgenommenen Ausrucksbewegung liegenden inneren Verhalten eines Anderen. . . . Ich freue mich mit demjenigen, dessen Freude ich in seinen Ausdrucksbewegungen sehe, ich erlebe sie unmittelbar in der Betrachtung der Ausdrucksbewegungen, wofern nicht Freude solcher Art meinem eigenen Wesen widerspricht. Ich tue dies um so sicherer, je mehr ich in der Ausdrucksbewegung in voller Freiheit, also unabgelenkt durch nebenhergehende Gedanken und Interessen, betrachtend bin und verweile. Und ich freue mich ebensowohl in der Wahrnehmung des fremden Zornes oder des fremden Schmerzes, wenn ich diesen Zorn oder Schmerz frei innerlich mitmachen kann, d.h. wenn in ebensolchem Zorn oder ebensolchem Schmerz mein eigenes inneres Wesen frei sich ausspricht."

Obviously, Lipps's explanation is based upon the James-Lange theory of emotion, which assumes that a feeling state is the awareness of organic sensations. We have shown before (Vol. I, Chs. 1 and 2) that the awareness of organic sensations can never add up to feeling. Rather, feeling is a secondary reaction to a perceived state of functioning, accompanied by various bodily changes of which we become aware. Lipps's theory has been criticized by, among others, Scheler (1923) and Edith Stein (1917). The latter acutely remarked: "I am not 'one with' the acrobat; I am only 'with' him." As Edith Stein implies, we are with the acrobat in imagination. We see the situation he sees, we know what needs to be done and make the effort he does. In the same way, the onlooker at the races is urging on the horse he wants to win. At the 100-yard dash, he holds his breath and braces himself for the athlete's desperate end spurt. So also, the back-seat driver steps on the (imaginary) brake when he thinks the driver should, not because he "feels himself into" the driver but because he sees the light turning red or another car cutting into his lane. What is called "empathy" is really a "living the situation" *instead of* or *with* the other; we experience the same emotion as the other, not because we imitate his expression but because we literally share his experience even though only in imagination. Scheler calls this "Nachfühlen" (vicarious feeling). We would prefer to call it imaginative participation or empathy.*

This living in imagination is unwitting, spontaneous, and requires only concentration on the situation as if it were *confronting us*. It happens even when we do not want to feel the emotion the situation arouses in us. There are theatergoers who are so moved by the hero's plight that they refuse to see a tragedy so as not to be exhausted by their emotion. There are people who confine their reading to detective stories that offer intriguing puzzles in detection but leave their feelings unmoved. The emotional participation in drama was considered a beneficial catharsis by the Greeks. When life is drab and does not seem to have room for a great love or a devotion to something worth while, the theater gives us a chance to be at our human best at least in imagination, for a short spell. The emotions aroused are genuine, as genuine as the emotions aroused in a dream. But as in the dream, they do not outlast the situation that gives rise to them. Only when the drama has touched us to the point where we are led to reappraise our routine convictions or attitudes, and change them for the better, will there be a permanent benefit.

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* There is extensive experimental literature on empathy which has little relevance to this phenomenon and will not be reviewed here. It will be discussed with other attempts at measurement in the Appendix.

Sympathy. According to Scheler, vicarious feeling eventually leads to fellow feeling. For him, fellow feeling ensures that the reality of another person is brought home to us; it disappears when the supposedly real person (or animal) turns out to be fictitious. Fellow feeling or sympathy presupposes that we understand the situation of another and his feelings in it; this is not true, as we have seen, of empathy or the love of nature as such.

We agree with Scheler that fellow feeling or sympathy presupposes understanding the other's situation and responding to his feeling. When a man has sympathy for another, there seems to be a fundamental agreement between the persons concerned which makes it easy for each one of them to appraise a problem, an issue, or a person as the other does. When we say we are in sympathy with a person or a group or a policy, we mean that we are like-minded and judge a problem as the others do. We have the same basis for appraisal, and thus our estimate of a particular situation and our emotion will be similar. It is not imaginative participation because the emotion need not be aroused at the same time that others feel it. In the widest sense of the term, sympathy seems to be the emotion that unites us with others who share the same existential position. It is a fellow feeling that unites us with other human beings and finally with all living nature, in so far as it is existing, living, and confronted with similar problems.

However, sympathy in the restricted sense of the term, as pity or compassion,* seems to include a reaction to the other and also a reaction to his emotion. Suppose a friend of mine has been told he has cancer. I understand his situation and see the possibility of long invalidism and a painful death as he sees it. As a result, I feel his fear with him. But I also appraise his fear as it affects me: he is dear to me and I want to comfort him and do what I can to help him; I surely feel sympathy. To have sympathy it is not even necessary to feel empathy. I may recognize his fear and may not let myself feel it with him; rather, I may turn away from it to encourage him and comfort him instead.

It may be argued, of course, that sympathy without empathy may produce a shallow Pollyanna attitude; that to comfort another effectively we must first feel with him, feel as he does, however briefly, before we can arouse a desire to fight the calamity, muster the courage that may be impossible for him who is completely immersed in his fear. Only when we have participated in his fear will we be able to comfort another effectively by giving him an opportunity for empathic courage. Ideally, sympathy may have to include empathy.

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* "Compassion" used to be, as "sympathy" is, a broad term but has been restricted to "pity." The root of both words is the same, *path.*

At any rate, sympathy includes a determination to do something about the other's plight. Sympathy as pity or compassion is always aroused by something harmful that has befallen another, it is an answer to another's fear, sorrow, distress. Sympathy takes sides; it is based on a judgment that another's suffering is bad and that we must help him to bear or combat it. Sympathy may be restricted to fellow suffering, when nothing else will avail, or include a deliberate effort to help.

Sympathy is akin to pity.* Whenever another's suffering is realized and appraised as bad, we feel sympathy or pity. So we feel pity for a child or even an animal that is cold or hungry or abandoned. We feel for a man who is abused or roughly treated and cannot defend himself. But the degree to which we can feel pity or sympathy will depend on the extent to which we can appreciate another's plight, as well as on our concern for him. No matter how warmhearted or affectionate a man may be, if he is insensitive or unimaginative, only the most obvious or the most intense suffering will arouse his sympathy. Only when he actually sees the ravages of disease or starvation or when he actually witnesses ill treatment will he be able to feel sympathy for the sufferer.

However, sympathy also implies a rational judgment. Unlike any other emotion, sympathy can always be avoided by turning away from the sufferer. If we ourselves suffer fear or distress, there is nothing we can do to escape it as long as the danger or pain continues. If someone else suffers and we appraise his suffering as bad and feel with him, we have sympathy, an emotion that urges to action like any other emotion. But as soon as we leave him or even turn our attention away from him, the emotion subsides. A rational decision is required to keep exposing ourselves to this particular situation, to continue feeling sympathy and so eventually be moved to action. Accordingly, Scheler insists that sympathy is ethical in its implications, while mere empathy (emotional infection as he calls it) is not.

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* The German for "pity" ("Mitleid") is a literal translation of "sympathy." In English, the emphasis seems to be on being sorry for another, pitying him, rather than suffering with him. "Sympathy" (like "Mitleid") is now used only in connection with human suffering.

Sympathy is not restricted to concrete situations. It can reach out to others even when we do not know them personally, and can extend to groups of people as readily as to individuals. Sympathy is felt more easily in a concrete situation because there is less demand on our imagination. On the other hand, another's danger can sometimes be better appreciated when it is described for us in all its implications and the plight of the victim is made clear in every detail. The attempt to rescue a child that has become wedged in a shaft, or to dig out miners cut off from the surface by an explosion, become tragedies of national importance as soon as they are reported in the papers, and are followed by extraordinary demonstrations of public sympathy. It is more difficult to arouse sympathy for a whole national group, but if the situation is serious enough, as during great floods or famines, imagination can easily supply the missing details and the sympathy of the public is immediate and generous.

Now how do these emotions help us in the pursuit of our self-ideal? As we have seen, outgoing love for everything around us is the basis for our feeling of confidence and belonging. Imaginative participation in situations confronting others affords us the possibility of understanding how they feel. Finally, sympathy makes it possible for us to overcome our self-concern and to come to the aid of others. Thus it expands our horizon and helps us take our place in the community of men.

Love and admiration. For Scheler, emotional identification is the basis for a more personal love. We have shown that it is basic only in the sense that a man must have the ability to reach out toward people and things, and that this ability finds its expression in a feeling of belonging. For sympathy, more than that is needed. A personal love for another surely is the first step to a love that embraces friend and foe, a love that can be called sympathy but is best known as *agape*. There is a progression in human love, as theorists from Plato to Scheler have recognized; and that love seems the highest that shows the lover at his human best.

There is admiration in every truly human love. Without admiration, love becomes bondage or addiction. Because he admires his beloved, the lover tries to make himself worthy of being loved in turn. For this reason, love and admiration are the prime movers in a man's progress toward a valid self-ideal. As with any other emotion, love also can be misused so that it serves a narrow egotism instead of enlarging the lover's heart. If it is excessive, it may occupy his mind to the exclusion of everything else. The religious man would insist that love tends to excess by its very nature unless it is moderated and balanced by a stronger love, the love of God.

In actual fact, our relations with others are based far more often on distinctively human sentiments than on instinctive tendencies or basic emotions. Human love is more than liking or sexual desire. The human emotions of admiration, sympathy, and love relate us to others, urge us to cooperate with them and wish them well. Just as intellectual, artistic, and other spiritual activities give more lasting delight than sense pleasures, so distinctively human emotions urge to more vigorous and effective action than do the basic emotions. In the Second World War, no appeal to future pleasure or immediate reward could have had the effect of Churchill's promise of "blood, sweat and tears." Man does not live by bread alone; he is most powerfully stirred by a person or an ideal that demands his full allegiance. Once he realizes that ideal as overwhelmingly important and eminently worth while, his effort to attain it is supported by all the emotional intensity of which he is capable, while a casual appetite, no matter how basic, is not. Spurred by devotion to such a cause, he may reach heights of courage and self-sacrifice that would be unbelievable if his actions were founded on sublimated libidinal urges or on secondary drives.

THE LOVE OF BEAUTY

As goodness arouses admiration, so beauty kindles delight. Beauty, like goodness, can be appreciated only by human beings; it has no appeal for animals. What is beautiful is admired for itself, not for any use it may have or any appetite it may satisfy. For the animal, beauty does not modify the appeal of what is wanted. A beautiful object is an object like any other; its beauty adds nothing to its use. For the human being, beauty is something over and above any use to which he may put a beautiful object. Beauty demands contemplation pure and simple, whether we find it in a vase, a painting, a landscape, a symphony, or a person. To possess beauty is neither having and holding nor having and taking. It is solely a looking and beholding.

A woman's beauty may be appreciated by her lover, but it is not created by love, and still less by sex. Freud thinks that the sense of beauty must "obviously" spring from sexual desire because for him the libido is the root of all human emotion. Yet he notes with surprise that the genitals, which arouse the strongest desire, are rarely thought beautiful. How sexual desire, aroused to greatest intensity by something not beautiful, could develop into an appreciation of beauty, he fails to say.

There is no doubt that the beautiful does give sensory pleasure. Music pleases the ear, paintings please the eye. But esthetic emotion is not sensory pleasure. What we enjoy in a work of art is its unique order and harmony, the human significance we find in it. When we contemplate a beautiful landscape, we see a harmonious pattern, we grasp the relation of mountains, trees, and meadows. We see them as a whole, belonging together, having a full and satisfying meaning. This meaningful composition we love and enjoy. Our joy completes us, makes us whole. The contemplation of beauty rises beyond mere sense and self-concern; it is a satisfying, perfective, and integrative human experience.

Such an experience arises from contemplating natural beauty as well as from enjoying a work of art. To produce a work of art, the artist applies his good taste and skill to making something that can provide such an integrative and perfective human experience. The objective of the work of art appeals to what is truly human, as does natural beauty. We admire order and harmony but also excellence in every sphere of life. Hence we admire the skill of the artist who works with love and care, using the right means for expressing what he sees.

It is no accident that the love of beauty and the love of nature awaken during adolescence when boy and girl become aware of human values and try for the first time to order their life deliberately in accord with some ideal. The unthinking feeling of oneness that was alive in the child's trust in people and things now becomes an appreciation of the beauty of nature even in its terror and majesty. The adolescent begins to admire excellence as seen in the harmony of all life, of all nature. The enjoyment of the beauty of the human body no less than the beauty of the beloved becomes a facet of human love, as does the admiration of the beloved's excellence as a human being. Such admiration and esthetic delight, and the worship and tenderness they bring, complete truly human love.

Lipps appeals to empathy for an explanation of esthetic emotion. For him, empathy is the means of understanding a work of art. But we can no more understand a work of art by empathy than we can understand human emotion by empathy. In both cases, we must first understand what is expressed, in a painting or in a human face, before we can feel the emotion expressed in it. Lipps may be right in claiming that the ***enjoyment*** of a work of art depends to a great extent on our ability to participate in the emotion it expresses. In contemplating the soaring spires of a Gothic cathedral, the exalted arch of the nave, our participation in the upward movement helps us grasp the meaning the artist intended: that there is something above man to which the spire points. The architect expresses in stone what we can feel in looking at his work. By imaginatively participating in this "prayer in stone" we are led to pray in reality. We do not grasp the meaning of the cathedral by becoming aware of the kinesthetic sensations felt by following the upward movement of its spires; rather, the upward movement makes it possible to imagine a striving toward something above man and so to feel the reverence and awe that accompany such striving.

THE JOY OF DOING AND MAKING

In our discussion of feeling (Vol. I, Ch. 4) we saw that activity is accompanied by pleasure and well-being. This pleasure is a basic psychological reaction, experienced by infants and animals as much as by adults. But as the child grows and develops, he begins to experience a joy in activity that goes beyond the analogous experience felt by infants and animals. While the infant's play is much like that of a puppy or a kitten, a joy in motion for its own sake, the child gradually begins to discover the joys of rhythmic motion. He learns to swim and to ski, but also to sing and to dance. Rhythm adds a new dimension to the joy of movement.

The child also progresses from handling things and using them in play to the discovery that he can change them. At first, the only way he can show his mastery is by breaking or destroying them. Later he finds that there is greater mastery in imposing a new pattern on the things he finds, whether by drawing, coloring, modeling, or building. Gradually he discovers that in making something he can improve on what it was before, that he can give expression to his idea of the thing. The joy of creative endeavor is for man alone, whether the result of his effort is a poem, a painting, a philosophical system, a piece of machinery, or a social enterprise.

When a man's creative effort is aimed not at making something useful but at making something beautiful, he experiences the extra joy of artistic creation which allows a unique self-expression. While the early pleasure in activity is a feeling, as we have defined it, the satisfaction that goes with making something is a joy in the achievement, in the intention realized, rather than a pleasure in the doing or the making. Indeed, the doing or making may be slow and laborious. The travails of creation are known to anyone who has ever tried to express an idea and give it tangible shape. It is the goal, the possibility of achieving what he aims for in artistic perfection, that makes a man keep on despite repeated failure or agonizing partial success, until his idea is realized in full.

MIRTH AND LAUGHTER

Psychologists have shown little interest in the origin of mirth and laughter, but philosophers have been intrigued by it. Darwin considered the smile a remnant of the animal snarl, disregarding the entirely dissimilar meaning. All he wanted to do was to show the origin of the facial expression; once that was established, his theory permitted it to be used for various purposes. Darwin, like many a later theorist, was committed to the assumption that everything found in man must have its earlier form in animals.

We are not committed to such a notion. In fact, it is our contention that there are certain emotions that have no equivalent in the animal, for they depend on a reflective rather than an intuitive estimate of the situation. This is eminently true of mirth and laughter. To be able to appraise anything as ridiculous, it is necessary to compare an actual occurrence with what was expected. In the most primitive kind of slapstick comedy where a gentleman in cutaway and top hat slips on a banana peel, or two men sling custard pies, the humor lies in realizing the incongruity of the situation: someone adult and dignified is reduced to a childish state, either voluntarily or involuntarily. This implies a conception of what adult dignity means and what is incompatible with it.

It is true, of course, as Bergson points out, that laughter is not aroused by deliberate reflection. It is the result of a judgment that has become immediate and automatic, yet presupposes conceptual thinking. As Bergson says, it is our long acquaintance with social life that leads to certain notions of how people ought to behave. All the forms of the comic mentioned by Bergson are reducible to the appraisal of some incongruity between what *is* and what could be expected. For instance, he states the difference between irony and humor thus: "Sometimes we state what ought to be done, and pretend to believe that this is just what is actually being done; then we have *irony*. Sometimes, on the contrary, we describe with scrupulous minuteness what is being done, and pretend to believe that this is just what ought to be done; such is often the method of *humor*" (1911, p. 127).

To laugh at incongruity, we must be detached from the situation. As soon as we feel with the man who has slipped on a banana peel, as soon as we realize that he may be hurt and need help, the humor of the situation is gone. Similarly, a lover does not use irony in declaring his love, unless he wants to leave a loophole for his self-esteem that might be shattered were his proposal taken seriously and refused.

Since laughter indicates such detachment from the human situation, it is humiliating to be laughed at. When someone laughs at us, is amused by what we say or do, we realize that he regards us as no more than a specimen whose antics he watches and enjoys. It is part of human dignity to want to be considered as a person, no matter what the circumstances. This seems to be the meaning of Bergson's notion that the function of laughter, provided by nature, is to intimidate the individual by humiliating him and so make him conform. (In passing, it might be observed that it is rather incongruous to credit a blind though creative evolution with such deliberate design.) Of course, a man cannot help but notice the effect of his laughter on the man who is its object; eventually, he may come to use laughter deliberately to humiliate others and so assert his power over them. Conversely, the man who is laughed at will realize the concealed attitude of the laugher and will take care to refrain from anything that can make him a laughingstock.

Laughter deliberately used (at least in the sense that its effect on the other is known) is not only a product of detachment but results in a feeling of power. The laugher, as Bergson says, is disposed to look upon another as a marionette of which he pulls the strings: Bergson continues: "In this presumptuousness we speedily discern a degree of egoism and, behind this latter, something less spontaneous and more bitter, the beginnings of a curious pessimism which becomes the more pronounced as the laugher more closely analyses his laughter" (1911, p. 199). The practical joker enjoys his power over others. The more he sees them as marionettes and himself as the marionetteer, the more will he be tempted to assert his power by a cruel pull on the strings. Conversely, the man who plays the buffoon has the subtle satisfaction of being thought a puppet while it is he who manipulates the puppet master. Both for the buffoon and the practical joker, the human relationship is perverted into a relation between a man and his plaything. Sometimes the detachment so obtained can be used to bring home a much needed lesson without fear of punishment. A clever court jester could air the most unpalatable truths and get away with it. It is a pity that modern methods of communication make it impossible to tell a jest from an insult, or we might still have the jester with us. Our political scene, domestic and foreign, might profit from it.

Since laughter humiliates those laughed at, it has a most salutary effect when we can laugh at ourselves, for it helps us to see ourselves as others see us. The sting it carries may urge us to do something about the incongruity we have spotted in our actions. A sense of humor is a great asset in our quest for the self-ideal, for it will correct our shortsightedness, reduce the intensity of our desires to their proper proportions, and promote the humility without which we would never strive for human perfection.

For Freud, not unexpectedly, mirth and laughter is a defense mechanism. It represents economy in the use of energy. Thus *the comic* represents economy of thought; *wit,* economy of inhibition; and *humor,* economy of feeling. This energy release is under the control of the ego and represents a regression to infantile omnipotence, encouraged by a permissive superego. Critics have objected that explanations in terms of energy release can hardly grasp the essentials of the matter. If economy of thought were the essence of the comic, mathematical equations should be excruciatingly funny. In reply to this objection, Flügel (1956) made the point that regression to a childlike playful attitude toward reality must imply economy of thought for it reduces the effort of maintaining oneself at the level of reality thinking. But this explanation, he added, "is perhaps hardly the most natural or obvious way of describing what takes place" (p. 723).

As a defense mechanism, laughter "takes its place in the great series of methods devised by the mind of man for evading the compulsion to suffer —a series which begins with neurosis and delusions and includes intoxication, self-induced states of abstraction and ecstasy" (Freud, 1928, p. 3). Thus laughter is not a positive reaction to an incongruous situation but a defense against suffering, forced by reality and encouraged by the superego. It is difficult to see what is gained by such explanations. They are an attempt to fit the various forms of humor and the fact of laughter into Freud's system. Either as defense or as aggression, laughter is only one of many possible methods. It may be so used, certainly. But what is there about laughter that distinguishes it from other methods of defense or aggression?

Only man can laugh; but animals seem to appreciate at least some forms of incongruity. Puppy and kitten as well as the human infant are intrigued by a ball of wool that darts erratically across the floor. But the animals' interest seems to be a reaction to what is unpredictable rather than really incongruous. Only the human infant reacts with laughter to his mother playing "peek-a-boo," to her disappearance and reappearance, which is not only unpredictable but absurd. As the animal matures, he may still be startled by something unexpected, but he is not amused as is the human adult. The animal can never become so detached from the situation that he can appraise something as incongruous. However, there is one form of merriment animals seem to share with us: the sheer joy of living, expressed in exuberant activity. The gamboling puppy, the dog romping with his master seem to have experiences that are not essentially different from those of the child laughing and enjoying himself at swinging, playing, or romping. But this is not humor; it is merely pleasure in activity (see Vol. I, Ch. 4).

Humor, like other positive emotions, helps us in our quest for the self-ideal. Of course, all these emotions help us only when we give them the right direction. The desire to know may be expended in avidity for gossip rather than the knowledge of human excellence; admiration may be extended to the strong and brutal rather than the powerful and benevolent. Esthetic emotion may become so specialized and overvalued that it substitutes for the desire for truth and the love of goodness. And wit may be turned against the good, the true, and the beautiful just as easily as against evil and corruption. In the end, it takes a deliberate decision to follow the self-ideal as it ought to be; only when that is done will positive emotions help us toward the perfection of personality.

RELIGIOUS EMOTIONS

The religious man would argue that the most powerful help will come from the emotion that is the human response to the experience of the absolutely good, true, and beautiful: the experience of God.

It is often assumed that religious emotion is the experience of the "numinous" (cf. Otto, 1917), that it is a feeling of reverence, awe, and submission. But reverence and awe are not the only emotions that can be felt toward God, nor is submission the only way in which man can relate to Him. God can be experienced and appraised as a stern judge, but also as a "father," "brother," or "bridegroom." Obviously, the use of these terms hints at experiences that can be described only by using analogies with human relationships. To take them as proof of "projection" or "sublimation" of impulses directed toward human beings would be to mistake their import. Without the actual experience of such emotions as they portend, it is easy to call them "projections" because nothing in one's own life corresponds to them.

We have argued that the basic emotions are felt analogously by infants and animals, because they are rooted in an immediate intuitive appraisal. As the child begins to judge reflectively, rationally, his experience changes, for his appraisal now includes aspects that before were inaccessible to him. His emotions develop and become subtler as the growing child and the adult add new dimensions of experience. Finally, when a man begins to be aware of the Divine, his heart enlarges and he now sees what before he had no eyes to see. Naturally, he will use human terms for his emotion that convey at least a hint of his experience, terms that will be understood by everyone who knows a human judge, a human father, brother, or bridegroom.

From the available descriptions (see Underhill, 1911) it would seem that the love of God may be the result of a vivid realization of His goodness, His love and concern for men. This may be reflective but may also be aroused by a direct experience on what could be called the spiritual level. Such an experience seems to be as direct as sense experience, but without any sensory content. It may happen in a moment, with full awareness, or it may take some time during which there is no awareness of the outside world (mystical ecstasy). This is not the place to examine such experiences in detail. Suffice it to say that they are experiences in which love as we know it in human life has become intensified and transformed, hardly resembling even the love of God that is aroused by meditation and reflection.

At any rate, it is clear that such love will also urge to action, an action often ranging far and wide, producing prodigious achievements, as the biographies of such saints as Paul, Francis Xavier, Teresa of Avila convincingly describe. As an aid to the establishment of a worth-while self-ideal, and its singleminded pursuit under the most trying circumstances, this love of God has no equal.

HAPPINESS

There is one more emotion that is truly human yet remains mysterious: happiness. It goes far beyond joy or pleasure, is something that we seem to feel in snatches though our very nature demands its permanence. Strasser (1956) has presented us with an excellent phenomenological analysis of this emotion. We can do no better than outline it in a brief summary.

Strasser finds that happiness is related to joy or satisfaction because it is a fulfillment; but it is different from satisfaction, pleasure, joy, or serenity because it is the fulfillment not of a particular expectation but of the whole personality. Like pleasure, it is felt when something loved and wanted is actually possessed; but it differs from pleasure in that its object is not a particular limited thing. Like joy, happiness is a taking possession, continually renewed, continually rediscovered; it differs from joy in that it is a reaction not to the first taking possession of some good thing but to enduring possession of perfection itself. Serenity and happiness both imply continuity, but serenity stems from a memory of pleasurable experiences and is based on the certainty that a man is close to what he enjoys. The happy man does not need such certainty because he experiences the actual presence of the good itself.

Happiness, says Strasser, is always a reaction to an overwhelming abundance, an infinity, of the good and the beautiful. Whatever can cause happiness is always seen as full of infinite possibilities, yet it always brings a specific kind of happiness: stage success means happiness for the actor, the presence of the beloved happiness for the lover. Whatever brings us the experience of happiness is seen at the same time as infinitely good; it represents an infinity in this particular form. Thus success for the actor means permanent achievement, union with the beloved means happiness ever after. Happiness contains a hint of eternity. Even when its source is strictly temporary (e.g., the victory of an athlete at the Olympics), it carries with it the perfection of a moment that is eternal in its significance. Such happiness consists in the integral possession of a limited unit of time, a crystallized bit of eternity, "simul et tota possessio," possession altogether, all at once.* A marriage, the birth of a son seem the beginning of a "vita interminabilis."

Next Strasser shows that in human life happiness is seen sometimes as *taking possession,* other times as *unlimited possession.* Men find happiness in contentment—but contentment is endangered by change and violence. Happiness may be seen as luck, happy chance; but this would imply that happiness is only for the darlings of fortune. Happiness could be seen as harmony; but harmony requires artificial props and defenses, and can be maintained only by an elite shielded from disturbances. Happiness may be ecstasy; but ecstasy is not the way of normal human living. Happiness could mean liberation from the world rather than the crown and perfection of life: such a concept of happiness expresses the elementary longing of mankind for peace and rest from eternal change and activity; but is happiness really thinkable as lack of activity, of life, of personal commitment, is it not rather an activity of knowing, striving, loving?

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* Cf. Boethius: "Aeternitas . . . interminabilis vitae tota simul et perfecta possessio" (Lib. V, 348, prosa VI).

For Strasser, these various types of happiness are all part of what happiness can be. Perfect happiness must contain all of these, must complement them, improve on them. Perfect happiness would have to reconcile what is contradictory in these experiences of happiness, would have to offer both harmony and ecstasy, both luck and contentment. If happiness can be conceived as liberation from the world, it has to transcend life in this world. Every kind of happiness actually refers to something that transcends the present, that refers to infinity in time and content. Perfect happiness must transcend all happiness that can be experienced here and now, it must be *Glückseligkeit* (happiness eternal). It would have to be ecstasy because a happiness that transcends present experience must necessarily be overwhelming. Yet, being perfect, it cannot be chaotic; it is the concrete presence of the good in itself and so must be a harmonious totality in which each person has a definite place, or the harmony would not be perfect. This happiness would be an ever new taking possession, ever renewed discovery, but also the security of enduring possession. Since, concludes Strasser, the possession of infinity is definitive, unique, nonretractable, happiness must be eternal, without duration, without sequence, without limit; the always young presence of the *simul et tota possessio*. Such happiness includes the joy of contentment, but also that of good fortune, depending on the way in which we use the unique chance we have been given in this life.

The religious man would say that this is a phenomenological analysis of the happiness promised when man sees his God face to face, and seeing Him will possess Him forever. Whether we accept Strasser's analysis of happiness or the description of it offered by religion, we must admit that the desire for happiness is one of the most puzzling features of human life. This desire for a perfection of joy that should last forever is a desire that is bound to remain unfulfilled in life as we know it. Yet it is the pearl of great price, the fata morgana just beyond the horizon, always hoped for and never reached. Like pleasure, happiness cannot be achieved by pursuing it directly, nor is it the incidental result of our own efforts. It is and will always remain a gift, to be given or taken away. Even such limited happiness as we do experience, for instance, the happiness of contentment, is always at the mercy of circumstances. At the same time, such happiness is at least an indication that we are on the right path, a token, the religious man would say, of what God has promised those that love Him.

The desire for happiness, even more than other positive human emotions, urges us to hold a steady course toward our self-ideal. Since the pleasures and delights in which we seek it disappoint us again and again, it urges us on and on. The man who despairs of fulfillment may become disillusioned, bitter, and cynical. But the man who sees it as a promise, the reward of love, may have a taste of it before he knows it and complete fulfillment in the end.

Appendix. PROBLEMS OF RESEARCH AND MEASUREMENT

Throughout this book, in both volumes, we have quoted research findings in emotion whenever they were relevant for our discussion. We shall conclude by discussing the problem of measurement in emotion and the way in which it has been approached in recent studies. In the last several years, there has been a growing interest in personality research which, either directly or indirectly, includes emotional factors. Often no mention is made of emotion in the findings though emotion is obviously involved: projective tests involve emotion, so does stress, frustration, or perceptual defense. It seems desirable to discuss some of these findings and point up their relation to a theory of emotion.

WHAT IS TO BE MEASURED?

Emotion is a fact of human experience. Since it is a fact, it must be possible to describe it accurately and measure it adequately. But thus far emotion, unlike sensation, has not lent itself to satisfactory measurement. Neither emotion nor sensation can be profitably measured as a momentary act. But because an emotional outburst is a rather spectacular departure from a person's usual behavior, it has been used as a unit in measuring "emotionality." In some nursery schools, for instance, parents are asked to keep a record of their children's development from birth on and to list all emotional outbursts, in the hope that these records will provide data for research. Apart from finding that such outbursts normally decrease as the child develops, these data have not yielded enlightening results. This is hardly surprising, for a mere counting of incidents discounts the context in which they occur and makes it impossible to see what role emotion plays in the child's beginning personality organization. Nor is the intensity of a given emotional episode worth measuring because intensity depends on the situation as well as on the person's appraisal of it. There is no reason to suppose that a man will always react with the same emotional intensity, no matter what the occasion, nor can we assume that every emotion will have a similar intensity.

If there is a genuine qualitative difference between emotions, we must not expect relevant results from studies of "emotionality" however defined, nor from studies of "tension" however attractive. Whenever significant results have been reported, the investigator did not find general emotionality (even though that may have been what he called it); rather, he found a specific emotional reaction to a specific kind of situation. Hall (1941), for instance, who succeeded in breeding "emotional" rats, described them as timid in open field situations. They were fearful and apprehensive to a much greater degree than the normal controls. There is no evidence that they showed greater intensity of any emotion other than fear. In fact, they showed considerably less anger and were less inclined to fight than their normal controls. In another study, Arnold (1942) found that "specific" muscular tension in the working hand accompanied high achievement in speeded-up shorthand writing ("effort tension"), but diffuse general tension went with low achievement. This diffuse general tension seems to have been the result of apprehension in a stress situation, although this conclusion was not brought out at the time.

Early in the history of experimental research, psychologists tried to discover "emotional" stimuli on the mistaken assumption that specific objects will arouse specific emotions, just as specific objects call out hunger or thirst or sexual desire. While some things were found that usually produced startle (see Landis and Hunt, 1939), and some that aroused disgust or dislike in many people (see Conklin and Dimmick, 1925), most emotions could not be aroused by simple stimuli, nor would a given stimulus arouse emotion in every person or under all circumstances. Once it was realized that emotion is a personal response to a situation rather than an automatic reaction to a stimulus, the attempt to demonstrate under what conditions emotion could be aroused was given up altogether. But such defeatism is not justified. If a specific appraisal brings about a specific emotion, as we have tried to argue (see Vol. I, Ch. 9) and there are well-defined conditions that will determine such appraisal, it should be possible to demonstrate this state of affairs experimentally. A situation appraised as promising satisfaction should arouse liking, one that threatens harm, dislike; if the experiment is so arranged that harm can be avoided easily, there should be aversion; if harm can be avoided only with difficulty, there should be fear.

To be certain that a particular emotion is actually experienced, we need the subject's report of the situation and how he appraises it. In addition, we have to know how he feels about it, that is, what emotion he can detect in himself. Granted that this report may not be as reliable as we would wish, it is more reliable than any single physiological indicator we could choose and is at least as reliable as the observation of such emotion by the experimenter. The experimenter's observation is often contaminated by theoretical bias. He may call the determination to succeed, or the worry over possible failure, or the fear of danger, indiscriminately "anxiety," because this is the term for which he has a theoretical explanation. Or he may insist that he cannot judge emotion because every emotion is simply a tension state, and so may content himself with listing various objective symptoms which are demonstrably inadequate as signs of emotion (cf. Hebb, 1945a).

PHYSIOLOGICAL INDICATORS OF EMOTION

Years ago, when emotion was investigated as if it were a simple sensory phenomenon, various physiological indicators of emotion were used in turn. The psychogalvanic reflex, heart rate, blood pressure, and respiration (both rate and inspiration/expiration ratio) were investigated on the assumption that emotion had some one physiological correlate that could be measured simply and easily. Such measures were even used as a more reliable sign of emotion than a man's own report. Where report and indicator did not agree, it was cheerfully assumed that introspection was unreliable.

Hans Syz (1927), for instance, found large psychogalvanic responses to the subject's own name, to sex words, to the name "Jesus Christ," but small deflections to "mother," "father," and to uncomplimentary terms (e.g., lazy, dishonest, etc.). The men reported emotion most often to "mother," or "father," but practically no emotion to words that produced the largest psychogalvanic responses. Syz concluded that their reports were unreliable because the galvanometer, after all, could not lie. Even if we could assume that the psychogalvanic response is a true measure of emotion, this conclusion would be unwarranted. Syz did not ask what kind of emotion was felt, nor did he consider that the shock of hearing taboo words (either sexual or religious) in an experimental setting might account for the deflection but would not be reported as emotion. Apparently, the subjects thought they had to report on the emotion they felt toward what was represented by the words, not on their feelings when hearing the words.

The psychogalvanic reflex (*PGR*). An example of the fate of such simplicist assumptions in research is the enormous number but insignificant outcome of the studies devoted to the PGR. After the initial discovery that there is a change in skin resistance with emotion, it was assumed much too quickly that the PGR is a simple physiological indicator by which to measure emotion. The fact that any kind of emotion produced a deflection seemed to confirm the notion that all emotions are accompanied by the same physiological changes. It was years before the investigators realized that not only emotion but also attention, mental work, and even mere sensory stimulation can produce a galvanometer deflection. When sensory stimulation is so intense that a waking animal would feel pain, a fall in skin resistance was found even when the animal was deeply anesthetized or decerebrated.

Further investigations finally clarified the physiological mechanism of the PGR. Apparently, the changes in resistance are the result of changes in the activity of sweat glands in the skin (not changes in the amount of perspiration). Since sweat glands are activated both by sympathetic and parasympathetic excitation, though the composition of the sweat differs, it seems to be established that the PGR indicates increased excitation of both branches of the autonomic nervous system (cf. McCleary, 1950). In addition, we have suggested on the basis of neurophysiological evidence (see Ch. 3) that the PGR accompanies any impulse to action, voluntary or involuntary, deliberate or emotional. The reaction to sensory stimulation, as well as startle and various emotions, are all accompanied by psychogalvanic deflections. These will be largest when there is a *sudden* impulse to action (e.g., in startle).

Since all action impulses, including emotions, are accompanied by psychogalvanic responses, the ambiguous results of studies in the PGR are easily explained. Sudden emotion is a strong impulse to action; hence the early positive results are explained. But startle is an even stronger motor impulse, which seems to account for the fact that the taboo words used by Syz produced such strong deflections. When a man's name is called unexpectedly, he is startled and gets ready for action—which explains why his own name usually brings about the strongest PGR of all.

Patterns of autonomic innervation. Now that many years of research have gone by, most psychologists are convinced that all attempts to isolate a simple physiological indicator of emotion are doomed to failure. The autonomic changes that occur in emotion are anything but simple. Any given emotion affects many glands and muscles, both voluntary and involuntary, depending on the action toward which it tends. At the same time, there is a homeostatic reaction that may start and finish at different times in nerves, muscles, or glands. The innervation of sweat glands seems to occur almost simultaneously with the experience of the emotional impulse, judging by the galvanometric deflection. In muscles and glands, the full effect may take some time to develop; the homeostatic reaction may not start until the emotion has disappeared. Thus there is a complicated picture of an interlocking series of changes which represent both the emotional effect and the organismic reaction (see Chs. 7 and 8). To correlate physiological changes with the emotion, we must either distinguish carefully between effect and reaction by repeated measurement during and after the emotion, or we must use as many physiological indices as feasible and show the total interlocking pattern of changes as they occur during emotion.

Lacey and VanLehn (1952) made a beginning with a variation of the first method, though they neglected to include the subject's report of his appraisal and emotion. Since they used mental and physical stress rather than emotion, the exclusion is perhaps not too important, although it is possible that the subject's attitude to such stress might influence the observed physiological changes. These authors demonstrated that different individuals react differently to the same type of stress. Some show the greatest change in blood pressure, others in heart rate, still others in PGR. But it is unclear whether these differences are the result of a different attitude toward the situation and hence of a different appraisal and emotion, or of differences in the reactivity of some organ systems as compared with others.

The most promising examples of the second type of measurement are the studies of Funkenstein and of Ax, also discussed in Chapter 7. These investigators measured a number of physiological changes during "anger out" (anger), "anger in" (self-blame), and "anxiety" (fear). These were genuine emotions aroused when the subjects found themselves unable to solve a series of difficult problems and the experimenter berated them for their poor performance. The physiological changes included changes in heart rate, ballistocardiogram, blood pressure, finger and face temperature, and the like. There was a clearly different pattern of physiological changes in each of the three emotions. "Anger in" showed the same changes as anxiety, only in milder form, while "anger out" showed changes in the opposite direction. Since some of the men characteristically reacted to ambiguous situations with anger while others tended to blame themselves and still others became apprehensive, these studies also demonstrate that there are habitual patterns of emotional reaction that are characteristic for a given individual. The typical reaction of neurotics was fear ("anxiety").

Biochemical changes in emotion. Of the many attempts to correlate biochemical changes with emotion, we will discuss only one of the more elaborate recent studies, that of Basowitz and associates (1955). Since it had been found in an earlier study that anxiety neurotics had a higher hippuric acid secretion * than normals (Persky *et al.,* 1950), the hippuric acid level was measured in a group of paratroopers in training. Such training is not only strenuous but usually arouses considerable fear. Contrary to expectation, the average output of hippuric acid was not increased in this group as compared to normal values, but it showed considerable variability during training. However, both amount and variability were less than was found in anxiety neurotics. Curiously enough, the self-rating of anxiety was negatively correlated with the hippuric acid level in the group as a whole (r — .67), though individuals with high self-rated anxiety showed a high hippuric acid level. Such a high negative correlation in spite of these exceptions means that in the great majority of cases low anxiety was accompanied by a high hippuric acid level. Apparently, there was a three-hour interval between the self-ratings, which were made on getting up in the morning, and the hippuric acid determinations. Consequently, Basowitz *et al.* suggested that anxiety was at a low ebb in the morning and had risen in the following three hours. Presumably, the men who rated themselves high in anxiety felt apprehensive all the time, even early in the morning. But there were also some men who rated themselves high on anxiety but actually had a low hippuric acid level. The same inconsistency was found in other biochemical indices.

The eosinophil level of the blood seemed to be the most responsive indicator of emotion. It was significantly lower after every training jump, even if a man refused to jump. Hence it seems to be the result of apprehension rather than jumping itself. Those who failed in their paratrooper training had a lower eosinophil level both at the beginning and the end of training than those who succeeded.

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* Hippuric acid is formed and secreted by the kidneys. It is increased after eating prunes, cranberries, and some vegetables.

Basowitz and associates found two kinds of anxiety in this group: harm-anxiety and failure-anxiety. Only harm-anxiety seemed to be related to the hippuric acid level and predisposed a man to failure. They concluded: "There is little question from our data that variations from the group means of many test measurements can be related to personality and its composite intrapsychic organization" (1955, p. 246). Yet men with schizoid personality, anxious and unstable adolescents, and men with comparatively high harm-anxiety passed the training course. Apparently, they had a dogged determination to succeed (as gleaned from interviews), and succeed they did.

This study seems to illustrate what we have tried to bring out in Chapter 9 of this volume: that emotion as such can either hinder or improve action, depending on a man's purpose and his self-ideal. If it is important for a man to succeed and he has the ability to do so, he will be filled with courage and overcome fear. "Failure-anxiety" (in the terminology of the investigators) seems to represent courage, not fear. There is always a possibility of failure, granted, but a courageous man goes ahead in spite of it. Thus Basowitz and associates found that "failure-anxiety" did not hinder a man's performance, but rather the reverse.

PSYCHOLOGICAL RESEARCH RELATED TO EMOTION

Since emotions influence rational appraisal and action, the judgment a person makes and the attitudes he develops will include emotional factors; personality tests of various kinds will tap emotional differences. But the individual differences in the emotions described or expressed will necessarily be less important for personality evaluation than the way in which they are integrated into action. It is not nearly so important to discover whether a man feels anger or fear or love than it is to find out what arouses his ire, what makes him afraid, and what kindles his enthusiasm; and finally, to see to what actions these emotions inspire him.

Emotion in the TAT. Such data can be obtained through questionnaire methods, like the MMPI or the Bell adjustment tests, which are sufficiently accurate for the diagnosis of maladjustment or neurosis. But to predict and, still better, to understand an individual's actions, to discover the motives behind his actions, projective tests are far more adequate. Thus the TAT in particular should be useful for inferring emotional attitudes.

Unfortunately, there has never been general agreement on how the TAT should be scored, or even on what should be scored. Murray's original (1943) scoring of "themes," which counts "needs" and "press" separately, is rather cumbersome. Since then, a number of scoring methods have been developed, most of which allow considerable accuracy in clinical diagnosis (see Shneidman *et al.,* 1951). As time goes on, the TAT seems to be used more and more impressionistically, in much the same way as dreams are used by various schools of psychoanalysis. This has led to a general disappointment in the TAT as a valid measuring device. Wyatt and Veroff (1956) give some of the reasons for this state of affairs:

> There is the glibness and the unabashed game of working up dynamic-sounding formulas while pretending to produce diagnostic insight. The reason for that . . . is to be sought . . . in the many untested and untestable assumptions peculiar to the subject. It follows also from the transfer of concepts from psychotherapy . . . to personality testing. What sounds daringly dynamic to the neophyte turns out in reality to be based on a vast misunderstanding of psychoanalytic propositions and on ignorance of basic literature. (p. 54)

How justified this criticism is can be demonstrated with any number of TAT studies. We will select only a very few to show how these "untested and untestable assumptions" influence the results.

Lindzey and Goldberg (1953), for instance, tried to discover sex differences in needs and motives. They followed Murray's assumption that the TAT reveals latent and overt motives, and the psychoanalytic assumption that needs frustrated in reality will be expressed in fantasy. A five-point rating scale was used to indicate to what extent needs were expressed in TAT stories. These needs included achievement, aggression, sex, abasement, nurturance, and "narcism" (*sic*). Lindzey and Goldberg assumed that men should express more aggressive, sex, and achievement needs than women because these needs are stronger in men and cannot be completely satisfied in reality. Women, on the other hand, should surpass the men in abasement, nurturance, and narcism needs. The results did not confirm all the hypotheses. To a significant degree, men surpassed women in aggressive and sex needs, while women surpassed men in abasement and nurturance needs. But the men showed greater narcism than women, the women higher achievement needs than men, though these differences were not significant.

Now there is a reasonable doubt whether a need will be expressed in fantasy only if it is not satisfied in reality. It is not the man who often is too busy to eat who indulges in fantasies about food, but the gourmet who eats well and spends considerable time hunting up new foods or thinking up new ways of preparing them. It is true that starvation will induce food fantasies; this is Freud's "primary process," which is spontaneous and unwitting. But it does not follow that satisfaction will suppress fantasy. Imagination is not only spontaneous but can be directed and trained. Interest in a topic will inevitably increase a man's imaginative preoccupation with it, for interest, as an attitude, is a habitual tendency toward imagination and action.

There is another problem, namely, whether the TAT expresses latent or overt needs. Most TAT scoring systems (including the ratings of Lindzey and Goldberg) perforce depend on the "manifest" content of the stories. Since the TAT, like the dream, is a product of fantasy, it is usually assumed to have a latent content as well. While the manifest content includes emotion and action, the latent content is represented by unconscious impulses or needs. In theory at least, the manifest content is a disguise for the latent content. That disguise becomes very thin in stories of murder, rape, or robbery, which makes it easy to unmask the unconscious impulse. As the "affect charges" of these drives, the emotions described in the story should allow an inference about behavior determined by such drives, as soon as the disguise is penetrated. Unfortunately for this assumption, it seems singularly difficult to predict behavior, particularly normal behavior, from a TAT that is interpreted in this way. Kelly and Fiske's (1951) exhaustive study which tried to predict clinical success from such TAT interpretations is a good example.

For most interpreters, only the hero's emotions are the storyteller's own. The storyteller, it is said, identifies with the hero of his story and so experiences the hero's emotions. Piotrowski (1950), however, and more recently Wyatt and Veroff (1956) argue that the emotions or needs of every story character are really the storyteller's own. But is it true that the emotions described in the TAT stories are or have been experienced by the storyteller? There are some stories that are obviously autobiographical, as confirmed by an inquiry after the test. In such stories, the storyteller's own experience is recounted; accordingly, the hero's emotions (but no others) are his own. There are also stories that concern someone else, either someone in the storyteller's family or a friend or neighbor; this also can be confirmed in the inquiry. In these stories, the hero's emotions are just as obviously not the storyteller's own but belong to the person he is talking about.

It is often argued that these stories would not be remembered and still less told unless a man had a reason for remembering them. But does that justify the notion that they are significant because the storyteller feels as the hero does? Surely not. A story told by a nun with every sign of emotion is an example:

(Card 12) This man is dying after a reckless and dissolute life. He had left his family and had violently turned against religion. Now he is about to die yet he still refused to have anything to do with his family, cursed his enemies and vowed he would die as he had lived. But suddenly next morning he asked for a priest, wanted to see his family and died in peace. The prayers of his family had been answered.

It turned out that this was the story of an event that had really happened in the family of a friend and had made a deep impression on her. But what impressed our storyteller was the fact that the dying man at long last realized his error and that the prayers of his family were answered in the end. Thus she felt with his family, yet the emotions of the family are not even mentioned in the story.

In such instances as those above, it is possible to check on the storyteller's own emotions and attitudes. But what of the majority of stories that are neither autobiographical nor biographical? Surely it is more reasonable to assume that they are like stories about someone else than that they are like stories about the storyteller himself. Very often, the hero has nothing in common with the storyteller: a child may tell a story about getting married; or an unmarried woman may tell a story about a young boy. We cannot even assume that there is an imaginative participation in which the storyteller takes the role of the hero and describes what he would do if he were in the hero's situation. If that were the case, prediction would be simple: the storyteller would do what the hero is doing, given the same circumstances. Everyone who has worked with the TAT knows that nothing is further from the truth. The only possibility left is that the storyteller reviews various actions in imagination; and that he agrees with or approves those that are rewarded in the story or have a favorable outcome, and disagrees with or disapproves those actions that are punished or lead to misfortune and difficulty.

Many TAT studies could be cited that confirm our analysis. Whenever themes are counted or emotional incidents rated, there is little correlation between TAT scores and behavior. But when the story outcome is taken into account and used to indicate what the storyteller endorses and what he rejects, the TAT will predict behavior. Thus Sanford and others (1943) found that the intensity of "aggressive needs," as inferred from the number of stories about aggression, bears no relation to the actual aggressive behavior of adolescents. In a later study, Mussen and Naylor (1954) suggested that these results applied only to middle class boys who are punished for aggressiveness. In their own study of lower class delinquent boys they found that outward belligerence and the number of aggressive stories in the TAT were related. When they counted the number of times the heroes suffered punishment or misfortune, they found that those boys whose heroes were punished for aggression showed restraint in their actions; but this was merely a trend which did not approach statistical significance.

Perhaps more attention to the import of the stories would have improved the correlation. For instance, Mussen and Naylor would score the story of a boy who gets into a fight and breaks his leg as "punished aggression." But the story may go on to recount how the boy learns to play the violin during his enforced leisure, and eventually becomes a famous musician. The import of this story is not that aggression is punishable but that punishment, used constructively, can lead to success.

When Mussen and Naylor compared belligerent and mild-mannered boys, they found that the former told many stories of aggression and few in which aggression was punished. The well-behaved boys had few aggressive themes and many stories in which aggression was punished. Apparently, the aggressive boys told stories in which hostile and belligerent heroes neither were punished nor came to a bad end. It is not surprising that a boy who tells such stories expects to get away with his own malicious and aggressive actions. His "acting out" is the result of his expectation, betrayed in the story import, that violence will go scot-free.

If Mussen and Naylor's TAT data were rescored to permit ordering the stories into categories according to the storyteller's inferred approval or disapproval of aggressive action (as shown by the story outcome), it might be found that the group which showed overt hostility also endorsed the aggressive actions of their heroes. This, at any rate, was found in a study done by Petrauskas (1958) on naval recruits. The men who had been confined in the brig at least twice, told stories in which the hero either escaped punishment or never suffered any ill consequences at all. Recruits who had never been in the brig told stories in which wrongdoing was punished and deserved punishment was accepted.

In the last analysis, the way in which TAT stories are scored depends on the investigator's theoretical assumptions. If he believes that "needs" determine action he will concentrate on identifying those needs (including the need of aggression), diagnose the intensity of the need expressed in the TAT, and perhaps, in addition, investigate the interaction of several needs (e.g., aggression and fear of punishment). But if the investigator thinks of emotions as action tendencies that lead to overt action only if the person permits or indulges them, he will be interested in getting a comprehensive picture of the kind of actions an individual approves or disapproves. By and large, we can be sure, the storyteller will permit himself those actions of which he approves and will try to avoid actions he knows lead to disappointment, punishment, or remorse.

TAT sequence analysis. The latter assumption is employed in the TAT sequence analysis (cf. Arnold, 1949, 1951). Petrauskas's study (1958) showed that this type of analysis can validly discriminate between offenders and nonoffenders in a naval installation. Other studies have predicted high and low achievement among high school students (in 39 out of 40 cases: see McCandlish, 1958), or have selected good and poor teachers (50 out of 50 cases: see Burkard, 1958), when the middle range was excluded. In the TAT sequence analysis, each story is summed up in the import, i.e., what the storyteller is saying about his life situation. This is a simple condensation of the moral of the story, without any interpretation. Each import, as indicated by story and outcome, may be either constructive or not constructive. It is constructive when the story import indicates that achievement is the result of effort, initiative, virtue, or the outcome of a definite plan that accepts limitation and adapts to circumstances; when loss, harm, or danger is overcome by positive action; when ill-intentioned action is punished, rejected, or renounced; when others are met with good will, good fellowship, or humor.

The emotions that occur in the story seem to have been conquered when negative (e.g., fear, anger, resentment, discouragement) and fostered when positive (e.g., love, humor, cheerfulness). It is a reasonable assumption that stories which express such constructive attitudes are told by people who really have such attitudes. It would follow that they have developed habits of self-discipline which make it possible for them to achieve in school up to and sometimes even beyond their ability. Similarly, the positive attitudes they have developed (enthusiasm, cheerfulness, interest in work and in people) make them good rather than poor teachers. Since the criterion for constructive and nonconstructive attitudes has emerged from comparing a sample of good and poor students, teachers and recruits, but agrees with what every clinician would call good or poor attitudes, we may assume that such attitudes do exist and do help or hinder an individual in his daily life.

The sequence analysis gives us a picture of a man's motivational pattern, his self-ideal in action. Thus it can be used to predict whether he will act constructively or not. The sequence analysis will also show areas of conflict where firm convictions have not yet been formed. The normal person will reveal in the sequence analysis how he is ordering his life, not merely what are the raw materials that are so ordered. In the neurotic, the sequence analysis shows the preoccupation with particular areas of conflict, and in the psychotic, the difficulty, even the impossibility, of acting effectively. It is a moot point whether a "deeper" analysis will improve prediction. What does it profit us to know the emotions that harass a man if we are unable to tell whether he is their plaything or their master? *

Emotion and the Rorschach method. It is usually assumed that the Rorschach method yields a picture of the basic personality constellation. Consequently, it should allow us to estimate what role emotions play in the individual's personality organization.

The *M* response (human movement), as scored in the Rorschach test, apparently has no direct connection with emotion. According to Rorschach's original definition as given by Klopfer and Kelly (1942), it is an indication of the richness of a person's inner life, his creativity, and his acceptance of inner promptings. But in a later book Klopfer *et al.* (1954) say that *M* responses indicate a far more complex state of affairs. They denote intellectual capacity and imagination which are inner resources

* According to Korner, prediction would require the solution of two problems: the first, to detect the variables at work in adapting need (as diagnosed from projective techniques) to reality; the second, "to find the secret of ego synthesis which probably consists of an organismic process involving more than the sum of the variables at work, and which possibly is at the root of all the clinical discrepancies" (1950, p. 625).

In these pages, we have tried to sketch the sequence from biological need to perception, emotion, and action. We venture to suggest that the TAT, interpreted in the sequence analysis, holds the secret of "ego synthesis."

that make for stability. They also imply a value system as the product of intelligent, imaginative activity which results in self-acceptance. Seeing human figures betrays a capacity for empathy with others; since this is a reflection of himself, a content analysis of these responses will give a clue to a man's self-image (1954, pp. 254–64).

The key to these interpretations is the fact that *M* is scored only when there is a *kinesthetic quality* to the response, that is, when there is some sign that the movement or the impulse to it is actually felt. This would mean that a human movement response requires motor imagination. Like most psychologists, Klopfer and his associates make no distinction between visual and motor imagination, yet the distinction is important because different cortical areas are involved. There is a difference between seeing an image and imagining a movement. The painter, for instance, must have a good visual imagination; the sculptor needs tactual imagination as well. But the dancer, and even more the choreographer, must have superlative motor imagination if they are to work out an expressive dance. This would explain why painters as a group do not give many *M* responses (see Roe, 1946), a fact that has troubled many Rorschach theorists.

A human movement response not only combines visual and motor imagination, it is an imaginative achievement far superior to that implied in giving an *FM* (animal movement) response. The animal form is extraordinarily varied in shape, embracing spiders, snakes, bats, and all kinds of birds and mammals. All that is needed is to recall various animals and compare them to a likely contour in the blot. But the human form is comparatively stable, allowing a difference in size but not in shape. Response possibilities are limited because there are not too many contours that resemble this particular form. To see it in action requires an imaginative knowledge of what a person can do, which makes a human movement response a highly complex process.

In addition, action always includes emotion. Whether a man sees bears dancing, waiters gossiping, or clowns having a good time clapping hands, the movement reported is an emotional expression rather than a deliberate action. Thus the *M* score represents a whole sequence of imagination, perception, appraisal, emotion, and action, as compared with a form response which includes imagination and perception, but simply reports the object as it is instead of going on to appraisal, emotion, and action. A form response represents imagination and perception restricted to the objective, cognitive level; while the human (and sometimes the animal) movement response carries the perceptual process through to appraisal and emotion which finally culminates in action. Thus the person takes a stand, commits himself, instead of leaving the imagined and perceived form out there, apart from himself.

If the movement is really felt, the *M* response indicates what the person is prepared to do, emotionally speaking. It indicates his emotional action tendency but does not show us whether he would actually give in to it. The *M* response implies that a man has attitudes, but not whether they are constructive or not. Even a Rorschach response that is as integrated as the *M* response cannot acquaint us with his goals but only with his emotional attitudes; and only a content analysis of such responses can give us the quality of the emotions expressed. No wonder that content analysis has complemented if not actually replaced the quantitative analysis for clinical purposes.

According to Klopfer and associates, *FM* responses indicate "an awareness of impulses to immediate gratification," which stem from instinctual or infantile sources and so are difficult to integrate into adult inner life. Apparently these impulses are felt but not necessarily acted out. If we remember that human movement responses also indicate impulses felt rather than acted out, we might weigh the possibility that even *FM* responses indicate that the movement is somehow felt, not merely seen; that these responses also require motor imagination, in spite of the lack of a specific "kinesthetic quality" in the response. Actually there is no reason why this should not be so. To recognize even animal movement and what it is about to accomplish, we must be able to imagine the action and remember not merely what it looks like but how it is done. In such responses there is less of an imaginative effort but as much of an emotional action tendency.

The *m* response (inanimate movement) implies the awareness of forces beyond human control and thus a certain helplessness. The presence of such responses seems to indicate tension but implies an effort to maintain one's values—unless there are too many *m* responses, in which case we may expect helplessness and serious difficulties. Hence the proportion of *M, FM,* and *m* is important. If *M* is either greater than or equal to *FM* and there are no more than one or two *m* responses, the impulse life is properly subordinated to the individual's values (see Klopfer and associates, 1954). Phenomenologically, this response seems to imply not an impulse to motion but a recognition of being moved. This will create apprehension ("tension") rather than impulses to action that bring pleasure. From this point of view, both *M* and *FM* responses indicate definite emotional action tendencies, while *m* responses seem to indicate a vague apprehension, a wish to avoid outside pressure against which the person is all but impotent.

Next in the psychogram are the shading responses: *K* (shading as three-dimensional diffusion, like clouds, smoke, etc.) and *k* (shading projected into a two-dimensional plane, e.g., X ray). According to Klopfer and associates, the *K* response expresses a formless free-floating anxiety, the *k* response anxiety with some outward control. Phenomenologically, there is a bare minimum of imagination in the response "clouds" or "smoke," and not much more in "X ray" because the three-dimensional expanse is completely formless. Whether imagination can be hampered only by emotion is doubtful; but among emotions only apprehension (anxiety) could do so. Why such anxiety should result from a "frustration of affectional satisfaction" as Klopfer and associates claim is quite unclear, unless this response, like the *c* response, is considered a reaction to texture.

The specific texture response (*c*), indicates "an infantile, undifferentiated, crude need for affection of an essentially physical contact variety" (Klopfer *et al.*, 1954, p. 271). This response also requires imagination, but in addition to visual imagination it requires not motor but tactile images. To see something as texture, one must imagine touching and feeling it. Touch can be either pleasant or unpleasant: soft, furry things are pleasant to touch while sandpaper and ice are unpleasant. Granted that pleasurable contact is connected with affection, it need not imply sexual desire. Harlow (1958) has shown recently that infant animals are comforted more by contact than by food. It is a justifiable inference that anyone who gives many texture responses is preoccupied with the desire for loving contact, or, more generally, for pleasurable contact of any kind. If his texture responses betray that he has a general expectation of unpleasant contact, it is a reasonable inference that he may be suffering frustration. But whether this reasoning can be applied to shading responses is rather doubtful.

The color response (*C*) is usually considered the specific indicator of emotion. Rorschach held that color responses show a person's capacity for affective rapport; Klopfer and associates suggested that they indicate emotional reactions to the impact of the social environment, "how the person actually meets an emotional challenge in a behavioral sense" (p. 278). Phenomenologically, we would have to explain why colored cards represent an emotional challenge or indicate affective rapport.

Colors are not only the most obvious feature of the world around us; they are also liked. Some people, for instance, comment that the colored Rorschach cards are pretty. Most normal people find them at least a welcome change after the gray and black blots. But color appeals only to those who have a certain openness to the world around them, who are neither harassed by deep-seated conflicts nor so beset by anxiety that they become oblivious to their surroundings. A deep depression abolishes normal interests, and also prevents color responses. The depressive patient is preoccupied with black, that is with things that hold no life, no zest, no joy.

The mere fact that a man gives any color responses at all seems to show that he is open to the world around him. But a reaction to the color only, without any regard to form, a pure *C* response (e.g., "blue and yellow things"), represents a simple association of color and color name, which implies spontaneous imagination rather than directed imagination. Vague or indeterminate form in a color response (*CF,* e.g., "coral rock") shows some imaginative effort, though this is cut short before a definite outline is found. But a response that combines color and form in a sharply seen object requires both imagination and intelligence. For instance, bears are black, brown, or white in the real world; to account for the pink animals in card VIII by describing them as "polar bears in the setting sun" calls for both ingenuity and imagination. Hence the difference between *C, CF,* and *FC* responses is a difference between a completely automatic impulsive reaction in *C* responses, a reaction that requires some imagination (*CF*), and a reaction that is not only imaginative but appropriate and even ingenious (*FC*).

Impulsive *C* responses might indicate that a man is inclined to respond impulsively to everything he encounters. And impulsive responses are emotional responses, for they depend on immediate intuitive appraisal. Without reflection or deliberation, they lead directly to action, even if that action is only the spontaneous *C* response. A person giving such responses will react with immediate liking or disliking to everything that offers, and will yield to his impulses without any thought. He will be a prey to many impulses but may also have emotional explosions, as Klopfer and associates imply. Pure *C* responses are found in preschool children, schizophrenics, and people with organic brain disease or brain injury. Small children respond quickly, unthinkingly, to everything around them but may on occasion show a "lack of emotional control, emotionality of an explosive, hair-trigger variety" (p. 285). The same is true for patients with brain pathology. They react with anxiety and confusion when they are unable to cope with a situation, but only rarely with violence. The schizophrenic does react violently, without any attempt at control, when his hallucinations or delusions urge him to action; but at other times his affect is flat.

Contrasted with the *C* response, the *CF* response implies some directed imagination. The impulsivity shown by *C* responses seems to be checked sufficiently to take account of the real situation. The *FC* response, finally, seems to denote a man's loving regard for everything the world offers, so as to respond to it with verve and imagination.

This brief analysis should be sufficient to show the particular genius of the Rorschach test. It is unique in that it taps systematically the psychological processes that are indispensable for adequate functioning. In testing imaginative functioning in every sense modality (except for auditory imagination) it allows us to see how the individual uses imagination to integrate emotion into attitudes. To appraise the dominant emotional coloring, however, the quantitative scoring should be supplemented by a content analysis.

Research in anxiety. In a 1950 review, Hanfmann pointed out that many recent studies on various aspects of personality involve emotion, in particular anxiety. She mentioned that there are two patterns typical for anxiety, whether the studies deal with motor, perceptual, or ideational functions: either a disorganization of behavior or tight and rigid performance. We venture to suggest that disorganization occurs when the planning of action is inadequate, that is, when motor imagination is interfered with, no matter what the reason. Tight and rigid performance, on the other hand, seems to indicate efficient motor imagination which inhibits impulsive expression and action.

Hanfmann concluded that the great promise of present methods of studying anxiety will not be fulfilled unless psychologists pay more attention to the way their subjects interpret the situation and to the emotions they feel. There is no doubt that such a change of attitude is necessary if we want to make further progress in the study of emotion. Once we realize that a man's emotional reaction absolutely depends on his interpretation and appraisal of the situation, it becomes imperative to ask him about it. We may not be able to check up on his report, but at least we have data we could get in no other way. It is time we realized that the notion that a man does not know what he feels, and thus his report cannot be trusted, is based upon a theoretical point of view which may be mistaken.

Anxiety and perceptual defense. In many studies on perception there is a similar concentration on the situation while the individual's reaction to it is left unexplored. As an example let us examine some of the studies on perceptual defense.

The history of this type of research has been ably summarized by Postman (1953). He and his coworkers found that "negatively valued" stimuli required a longer tachistoscopic exposure before they were recognized than "positively valued" stimuli. Negative and positive values were found by letting a group judge to which of Spranger's six types of value a given word belonged. Words that belonged to the area valued most by an individual were called "positively valued"; words belonging to the area valued least by him were called "negatively valued." Postman and his associates concluded that a "perceptual defense mechanism" prevented the recognition of negatively valued words. Since psychogalvanic responses to the crucial words indicated an emotional disturbance even before recognition, they thought that this defense was repression. But they asked: "One may inquire at this point: 'How does the subject know that a word should be avoided? In order to repress, he must first recognize it for what it is.' We have no answer to propose" (1948, p. 152).

From the fact that autonomic reactions (PGR) to taboo words occurred before recognition, McGinnies later argued that this implied an emotional disturbance which delays recognition, while Postman suggested that the difference in recognition threshold may be the result of a difference in the familiarity of taboo and neutral words, or the result of a "set" for neutral words. He found that taboo words were more easily recognized than neutral words when the neutral words used were no more familiar (as determined by the Thorndike-Lorge word count). When subjects were told that failure in recognizing taboo words implies some emotional disturbance, these words were recognized even more quickly. Hence Postman concluded that there are specific perceptual hypotheses, "predispositions of the perceiver to organize stimulus cues in specific ways" (1953, p. 300). Thus subjects may at some time be inclined to expect taboo words, and at other times not to expect them.

It is not quite clear how these perceptual hypotheses arise. The set, as prescribed by the instructions, will certainly influence the individual's expectation. But there seem to be personality factors in addition to the prescribed set that may determine what he sets himself to see. Eriksen (1951a) found, for instance, that some people recognize taboo stimuli more quickly than neutral ones, while others recognize them significantly more slowly; yet the whole group had the same set, for they had all been warned to expect "unusual" words or pictures. Eriksen concluded that some people show "perceptual sensitization," others "perceptual defense," but both types of reaction are defensive in nature.

This raises the problem of why some people react with defense, others with sensitization. Eriksen (1951b) found that men who showed disturbances in word association (increased association time, failure to respond, blocking, etc.) also had a long recognition time for pictures showing scenes in the same area, for instance, aggression, homosexuality, succorance. On the other hand, men who quickly recognized aggressive scenes gave more TAT stories with aggressive themes than did others who took a long time to recognize aggressive scenes on tachistoscopic exposure.

According to Eriksen's (1952) own hypothesis, acceptable needs are expressed in TAT stories while unacceptable needs are repressed. But on this hypothesis, those who told many aggressive stories could not have repressed their aggression. They showed no disturbances in this area in the word association test and recognized aggressive scenes on the shortest possible exposure. This surely indicates that aggression is no threat to these subjects, does not create disturbance, and is not repressed. On the contrary, there seems to be a definite preoccupation with the topic. Hence we cannot agree with Eriksen that perceptual sensitization is merely another type of defense, "intellectualizing."

In a personal communication, Eriksen (1955) suggested that intellectualizing is not itself a defense mechanism. Rather, "certain kinds of defense mechanisms, for example, projection or reaction formation, might lead to perceptual sensitization for material that is basically repressed." But intellectualizers ruminate on sexual or aggressive topics and freely talk about them (Lazarus *et al.,* 1951). To do so, the material must be fully conscious and cannot have been repressed. Without repression, why any defense mechanism, whether projection, reaction formation, or intellectualizing?

Both sensitization and perceptual defense seem to be characteristic ways of responding to taboo words. Stein (1953) found that both "sensitizers" and "defenders" use their typical mode of reaction with different (but equated) sets of words. If they temporarily abandon it, they go back to it under pressure; when they are told, for instance, that the test usually indicates how well people get along together.

In another study, Lazarus, Eriksen, and Fonda (1951) found that "intellectualizers" are significantly more accurate in hearing and understanding statements with aggressive or sexual import against a background of white noise than are repressers. They also expressed sexual or hostile ideas more freely in the sentence completion test. In clinical interviews, the intellectualizing patient talked readily about sexual and emotional problems and ruminated on sexual and aggressive themes, while repressers blocked on such material and refused to talk about it. This surely shows that sexuality and hostility have a considerable attraction for the "intellectualizer." If there is a threat at all, it is overshadowed by the attraction. In clinical practice it is a truism that patients who talk a great deal about sex (even about their fear of sex) are really fascinated rather than frightened. The intellectualizers who show perceptual sensitization do not react with a mechanism of defense to a threat but respond avidly to a favored area. In Freud's time, people seem to have been afraid of their libidinal or hostile impulses, but today a good many seem to be quite willing to enjoy them.

That perceptual sensitization is the result of pleasurable preoccupation is also suggested by the results of Aronfreed *et al.* (1953). They found that unpleasant words required significantly longer exposure time for recognition than neutral words. Pleasant words were recognized slightly more quickly but the difference was not significant. Since none of the words used in this study touched upon the sexual area, we could perhaps not expect the ease of recognition that would betray intense preoccupation. But taken together with the earlier studies of Postman, Bruner, and McGinnies which showed that positively valued words are recognized easily, it does seem as if what was favored and valued was recognized quickly. Thus perceptual sensitization seems to reveal an interest in the crucial area while perceptual defense seems to imply lack of interest, dislike, or fear.

The mechanism of perceptual defense and sensitization. Granted that defense and sensitization are two different mechanisms and stem from a different origin, the real problem remains: How can fear delay recognition and interest hasten it? Ordinarily, something must be recognized before it can be either liked or disliked. Some theorists have suggested "precognition" or "subception," meaning an unconscious recognition which may be either admitted to awareness immediately or detoured and delayed. But to say that the recognition is unconscious does not explain why it is either speeded into awareness or delayed.

On the basis of our discussion of brain function we can offer a simpler explanation. Our discussion of emotion has shown that we not only see something but appraise it as good or bad and thus like or dislike it. We also appraise it for action. This intuitive appraisal initiates an impulse to action that is mediated by the action circuit; when neural relays via this circuit reach the premotor and prefrontal areas (areas 6 and 8), we experience an impulse to action. The action circuit also relays neural impulses to the motor cortex initiating emotional expression and physiological (autonomic) changes; it connects with the diffuse thalamic system and so modifies the electrical activity of the brain during emotion. Fear reduces the alpha index and produces low amplitude fast beta waves, while impulsive emotional reactions seem to produce slow theta waves. From experience we know that fear retards mental processes. From the fact that it superimposes its fast frequency gradually upon the whole cortex we gather that such retardation seems to be the effect of its interference with normal electrical activity.

The sensory impulses from the retina are registered no matter what a man's feelings are: there is an electrical deflection in the sensory cortex on stimulation even during anesthesia. But seeing or recognizing something requires more than sheer registration; it requires a vital reaction, namely, the experience of seeing, remembering, and appraising, mediated via the circuit from sensory cortical areas to hippocampus, amygdala, association cortex, and limbic system.

We have argued that every emotion leaves us with the expectation that similar situations will affect us similarly. Thinking and daydreaming about a particular topic is bound to strengthen this expectation, while fear and aversion have the opposite effect. The latter create a disposition to turn away from any topic that may become dangerous. When pictures of scenes are shown that either arouse interest or threaten harm, they are identified by remembering similar scenes that have had a similar effect. The intuitive appraisal that this is interesting or dangerous is based on the expectation that this scene, resembling pleasurable or dangerous situations in the past, will affect us in the same way. "Precognition" seems to be the intuitive judgment that here is something that is either wanted or feared. If it is wanted, the self-initiated process of exact identification is accelerated and the picture or word is recognized. If it is feared, the emotion (or rather, its effect on brain activity) interferes with the process of recognition. Thus we assume that the memory of weal or woe serves for a first approximate recognition and appraisal, which either helps or hinders the final identification. This hypothesis would explain why neutral pictures that resemble aggressive scenes (for instance, two people waving good-bye) often have similar recognition times as do aggressive pictures, whether the subject reacts with "perceptual defense" or "perceptual sensitization" (Eriksen, 1951).

The report of Aronfreed and associates (1953) also supports this hypothesis. They found that women were slower than men in recognizing all words, whether pleasant, unpleasant, or neutral; women also had larger psychogalvanic responses. It is not too far-fetched to suggest that women are generally more self-conscious than men and thus showed more apprehension in the experimental situation (as indicated by larger PGR). Accordingly, they took longer to recognize any of the words. When they were shown a sample of the words used, their PGR for all words decreased, apparently because their apprehension was reduced. When men were so informed, their PGR hardly changed, evidently because they had little apprehension to begin with. The men had a smaller PGR before they recognized unpleasant words, but a larger one just before they recognized pleasant words. Apparently pleasant words aroused a stronger action impulse on intuitive appraisal than unpleasant words. But for women, recognition and report itself seem to have increased the emotional action tendency, for their PGR always increased right afterward. Even before recognition, their PGR was much higher than the men's, which seems to account for the interference of emotion with accurate identification and which made for slower recognition. Since the women were not asked for a report of their subjective experience, it is impossible to say whether this explanation is correct or not.

At any rate, it becomes clear that in this type of research a careful analysis of the subjective state of "sensitizers" and "defenders" (or whatever we may choose to call the two groups of people) is essential before we can come to any satisfactory explanation of these two types of reaction. If fear interferes directly with mental (and brain) activity, as seems likely from our evidence, it is unnecessary to postulate repression as a defensive mechanism. If interest and liking facilitate such activity, perceptual sensitization cannot be called a defense.

Studies on "empathy." In recent years, there has been a flurry of studies on empathy which subsided almost as quickly as it flared up. As Cronbach said, empathy (like identification and projection) was regarded as an "intuitive, pervasive feeling state which preceded any cognition" (1958, p. 376). Dymond (1949) who started this particular research interest, defined empathy as "the imaginative transposing of oneself into the thinking, feeling and acting of another and so structuring the world as he does." She referred to several recent suggestions that empathy may be essential for our understanding of others, including the psychotherapist's understanding of his patient. In this formulation, empathy undoubtedly involves appraisal and emotion.

To measure empathy, Dymond asked students to rate themselves on the following traits: self-confidence, superiority, unselfishness, friendliness, leadership, and sense of humor. Next, each student had to rate the others in his group as those others would rate themselves; and also, to rate himself as he believed the others would rate him. Dymond thought that empathy must be involved in the judgment of how others would rate themselves because the judge must "transpose himself into the thinking, feeling and acting of the others. If he can do this he should be able to predict how the others will behave in certain defined situations. The situation chosen to test this ability was the subject's ability to predict how others will rate themselves and how they will rate him on these six traits" (1949, p. 128).

This looks at first glance like an ingenious way of solving the difficulty of rating traits. If one person can "transpose himself" into the feeling and acting of another, why not transpose himself into the other's activity of rating? But here an obvious difference is glossed over by a metaphor. We do not really "transpose" ourselves into somebody else's activity. We take another's place imaginatively and look at a situation the way he would look at it. To look at the *situation of rating himself* in such an imaginative way is a much more difficult feat than, for instance, to feel with him as he is hurdling a fence.

Our discussion of empathy (Ch. 11) has shown that empathy in the strict sense means that we feel an impulse to move in the same way another person moves because we see ourselves in the same situation as he.

There is also an imaginative identification with another that is deliberate. But in this case, we imagine what the other would do out of our intuitive and reflective knowledge of him. As role theory would have it, we try to play his role as worker or professional, as Democrat or Republican, as good father or stanch friend. Our judgment of what he would do in any of these roles is only as good as our knowledge of him. We appraise another as being pleasant or unpleasant, as being congenial or as having nothing in common with us. We know him as someone we like or dislike, who is friendly or cool toward us. We also try to discover his motives and to understand the reason for his actions so that we come to know what he would do on various occasions. Both our intuitive and our reflective knowledge of another are influenced either directly or indirectly by our own system of values and, not least, by our favorite system of psychological interpretation. A psychoanalyst and a nonanalytic psychologist may ascribe quite different motives to the same person and thus predict very different actions.

A prediction of how another would rate various traits (whether they are his or someone else's) depends on how well we know him and how adequately we can infer his picture of himself and of others from our knowledge. These two features are not necessarily related and should be judged separately if we want to know something about the way in which we appraise others and judge their motives. We shall see from a brief review of some studies on empathy that these two factors are neither recognized nor controlled.

In Dymond's original study (1949), the students who judged each other's self-ratings were divided into several groups of six members. Each group met once a week to discuss and plan a class project. One group was congenial from the beginning. This group had the highest "empathy" scores on the first test (given after they had had three meetings) and improved still further on the second test (after eight meetings). Another group engaged from the beginning in personal feuding. Its members had a poor empathy score on the first test and a still poorer score on the second test. This surely shows that people who like each other come to know each other better than people who dislike each other.

Dymond also found that the women improved their scores on the second test while the men did not. Since men and women were evenly distributed over all the groups, and the first test showed no significant difference between men and women, this seems to mean that further acquaintance improves women's predictions but not men's. It is unlikely that women are more adept than men in making logical inferences. At any rate, there is no evidence for a feminine superiority in reasoning. But it is generally conceded that women are more interested in people while men are more interested in ideas, in other people's convictions and interests. Thus Dymond's findings would mean that women get to know other people better than men do and for this reason are able to make better predictions. What Dymond seems to measure primarily is a person's knowledge of another, what could be called "understanding another," in the sense of the German *Menschenkenntnis.* This is far removed from empathy proper, for which it is sufficient to see another in a situation in which we can participate.

Later, Dymond devised other situations to measure empathy, for instance:

(1) Children interpreting a picture of various episodes by describing the emotions of the characters (Dymond *et al.,* 1952)
(2) Children judging which classmates like and dislike them (Dymond *et al.,* 1952)
(3) Nondirective counselors predicting which statements their clients will endorse and which reject in a Q-sort (Dymond, 1953)
(4) Marriage partners predicting each other's answers to MMPI items (Dymond, 1954)

Now let us see what is measured in these studies.

(1) To mention various emotions that might be felt by people in a picture is merely an indication that a pictured social situation can be appraised in some way. Since the score employed is "based entirely on how readily these [emotions] are elicited by the stimulus materials and the tester's prompting" (Dymond *et al.,* 1952, p. 205), the situation need not even be understood, the child may simply confabulate.

(2) A child's recognition that another child likes or dislikes him is a direct intuitive appraisal of the other's friendly or hostile behavior. But it is also affected by the child's own feelings for the other. If he likes another child, he may interpret the other's disinterest as liking; if he dislikes a child, he may interpret the other's casual friendliness as irksome affection.

Since there is an uncontrolled factor in both these studies, it is not surprising that no correlation could be found between them, either for second-grade or for sixth-grade children. Neither of the measures used is a measure of empathy. The score in the first study shows merely that the child can ascribe emotions to people in a picture—not whether he can interpret emotions correctly. The score in the second study indicates how well a child can recognize another's like or dislike of himself, and even this appraisal is contaminated by the child's own emotion. If the picture interpretations were rescored for correctness or at least reasonableness of the assigned emotions, we would expect a positive correlation between a child's recognition of emotion in pictures and his recognition of other children's feelings toward himself, provided always that the pictures portray the social situation as well as the emotional expression of the actors. If a score could be devised that would control the factor of the child's own emotion in rating the feelings of others, the correlation should be increased.

(3) The counselor's prediction of the client's Q-sort, like the prediction of others' self-ratings, depends on understanding the client and making the correct inferences from such acquaintance. Of course, the counselor has an incomparably better opportunity of getting to know the client than had the students in Dymond's first study. The correlations between counselor's prediction and client's self-sort ranged all the way from .05 to .84. Dymond concluded that some counselors can make valid predictions and implied that these counselors had more "empathic" ability. What Dymond measured here is not empathy; but neither is it true that the clinicians made *"statistical* predictions in an informal, subjective and uncontrolled way" as Sarbin (1944) says of clinicians' understanding of their clients. In this situation, the clinician knows his client and has to judge the client's self-knowledge. His knowledge of the client is both intuitive and reflective, his judgment of the client's insight is inferential; and inference and reflection are based on *significant* signs, not on the number of signs. Logical inference is not statistical prediction.

Predicting a marriage partner's MMPI likewise depends upon a knowledge of his person and his reactions in various situations. As we would expect, happy couples were found to be significantly more accurate in their predictions of each others' answers than were the partners of an unhappy marriage. The latter assumed significantly more similarity between their spouse and themselves than was warranted by fact. Happy couples gave more similar answers to the MMPI but did not deceive themselves about their differences. Here as in Dymond's first quoted study, the findings indicate that mutual liking improves mutual understanding. Such understanding is not the result of "empathy" (i.e., putting oneself in the other's place and looking at things as he does) but merely of loving him and knowing him.

Other investigators have pointed out that modern research in empathy has little relevance to empathy as originally defined. Snygg (1953) and Luchins (1953) suggested that recent research is really an attempt to measure social interest or understanding. Luchins, particularly, thought that such understanding depends upon the self, the other, and the objective situation, and that we must first find the conditions that favor understanding before we can try to increase it.

Many later workers measured aspects of personality that have only the most tenuous connection with empathy. Speroff (1953) measured "mass empathy" by tapping a person's social information (his knowledge of general preferences in buying magazines, listening to music, and the like). Fiedler (1953) measured "assumed similarity," the efficiency with which a person can predict the reactions of others on the basis of his own. Other studies of social stereotypes are even farther removed from empathy. They are irrelevant for our purpose because they have no connection with emotion or intuitive appraisal. The ratings they require can be made without an immediate acquaintance with other people. The individual is simply asked to indicate the probable tastes, attitudes, or preferences of a given social class, sex, or age grouping, and does so on the basis of what he happens to know of these groups.

Such studies ask for an inference, logical or not, from a diffuse kind of knowledge that on occasion may border upon ignorance. This is a far cry from investigating the "empathic ability" Dymond had in mind. A careful analysis of the process would have prevented much confusion and would have produced results that are a permanent contribution to knowledge instead of the doubtful and ambiguous findings that represent the sum total of a prodigious research effort. If we decide to make a clear distinction between *empathy* as the felt impulse to move as another does, *knowing or understanding another* as the intuitive and reflective appraisal of his person, and *predicting his actions,* based on an inference from our knowledge of him, we shall be able to investigate these factors separately and come to valid conclusions.

Emotion in "person perception." In recent years, the intuitive and reflective appraisal of others has begun to be investigated systematically. From the original studies on empathy, research has moved into the large field of "person perception" (see Tagiuri and Petrullo, 1958). It is partly linked with cognitive theory, partly with social psychology. We propose to mention only a few facets of this research which are related to emotion.

Horwitz (1958) investigated liking and disliking of other people. To explain this phenomenon, he assumed that there is a need system which arouses these emotions. He showed experimentally that liking can be aroused when a situation occurs that can be perceived as enhancing one's power; disliking can be aroused when the situation can be seen as reducing one's power. Horwitz does not say but seems to imply that all liking or disliking is aroused in this way. He has to postulate a need system because he, like most psychologists, does not think of emotions as action tendencies but as the experience of a drive or need.

Now it is obvious that we like things and people not simply because they increase our power, though for some people and in some circumstances this may be one reason for liking them. Even Horwitz's experiment admits of other interpretations. He established two classroom situations: one teacher-centered, in which it was understood that any decision was to be made by teacher and group, but that the teacher's vote would have double the weight of the students'; another student-centered, where it was understood that the students' vote would have four times the weight of the teacher's. Both groups were asked to make five different things. In the middle of the instruction on how to make each object, the teacher asked for a vote as to whether he should repeat the instructions or go on. In all cases, the students voted for a repetition, but always the teacher chose to continue and did so. All the students were indignant, but those in the student-centered group much more so than those in the teacher-centered group. Since the power of the student-centered group was reduced much more drastically than that of the teacher-centered group, Horwitz concluded that it was this power reduction that increased the students' dislike of the teacher. In other experiments, students expressed a liking of the teacher if he either conformed to the rules or acted in favor of the students, overriding the rules. Horwitz inferred that this procedure enhanced the students' power and generated a need to like the teacher.

If Horwitz were right, observing the rules should not arouse liking but merely an absence of dislike, for the students' power is neither enhanced nor reduced. Yet fairmindedness itself, as Horwitz pointed out, results in liking the teacher. It is more reasonable to suppose that the teacher is liked because he gives everyone his due and more than his due;

that this enhances the students' power is incidental. Some students, and apparently the experimenter, may prefer this explanation, others may not appraise the situation in this way at all. According to our theory, what is appraised as good is liked; and fairmindedness or giving in to the students' decision certainly was so appraised.

That liking and disliking do not depend on a "need system" is shown also in a study reported by Newcomb (1958). Seventeen students, unknown to each other, were given a questionnaire on which they had to indicate a favorable or unfavorable attitude to a hundred items, "from President Eisenhower to classical music, from immortality to sexual morality, from house rules to university regulations" (p. 183). Those who expressed similar attitudes were attracted to each other as soon as they came to know each other, that is, after four months of living in the same student house. In the first few days of their acquaintance, however, men were drawn together who had quite different attitudes. This surely indicates that liking develops on the basis of mutual preferences because there is a similarity of convictions and attitudes; the liking does not create the similarity.

Now the early attraction cannot have been the result of a similarity of attitudes, but neither is it likely that these students enhanced each other's power immediately on meeting each other. It would be a reasonable inference that the early attraction was the result of an intuitive appraisal influenced by similarities with people liked in the past. Gradually, this intuitive appraisal was corrected by better knowledge and reflective judgment. To argue that there must be a "need system" before liking can be felt is arguing from a theoretical assumption, not from facts. No experiment can either prove or disprove such a notion. But we have tried to show that our alternative explanation is more economical because it can account for the facts without using *ad hoc* hypotheses.

CONCLUSION

This appendix was not intended to give a comprehensive review of modern research touching on emotion. Rather, it was meant to show on the basis of a few examples that research does not profit from ignoring emotions in favor of variables taken from various personality theories. Nor will studies of emotion give clear and unambiguous results if emotion is linked with a system of needs or drives in which it is at best an uneasy afterthought. Neither specific objects nor specific situations necessarily arouse the same emotion every time they are experienced; only when emotion is seen as the result of subjective appraisal can we explain such variation.

Research in emotion allows several approaches. There is, first, the possibility of *demonstrating a new phenomenon.* This could be compared to new discoveries in other sciences, though perhaps on a more modest level. The phenomenon is usually new only because it is either not experienced under ordinary circumstances or is not reflected on. Perceptual sensitization and defense, for instance, are such new phenomena, and so is the demonstration that memory and imagination are not unitary, global functions but distinct for the various sensory modalities (see Ch. 2).

Second, the *conditions* may be investigated that are required for a specific appraisal and emotion. Under this heading belong studies on anxiety aroused in various situations and under various conditions. Unfortunately, they usually do not include the person's report of the situation, of his appraisal and emotion. Thus "anxiety" remains a term which indicates the experimenter's preconceptions rather than the subject's experience, Some of the recent studies of interpersonal perception investigate the conditions of liking or disliking, but a great deal remains to be done.

Finally, we can investigate *the way in which emotion is integrated in personality organization.* Psychological research has always begun by investigating simple processes or functions. It has abandoned this approach only when it has become obvious that the most accurate knowledge of single functions will not allow us to predict human actions. We have always known that some people are more intelligent than others, or more purposeful and energetic. In measuring intelligence, our first attempts were unsuccessful because psychologists measured single functions: sensations, reaction time, memory, and the like. It took the genius of a Binet to recognize that intelligence must be measured not as if it were a single function but as it is expressed in intelligent actions. Because they have combined a number of situations that sample a person's performance, intelligence tests have succeeded where tests of reaction time or memory have failed. Intelligent performance presupposes memory, imagination, sensation, and it takes a certain time; but instead of measuring single functions, intelligence tests measure the directed use the person makes of them and his speed in doing so.

With emotion also, psychologists tried at first to explore it as a single function by investigating objects or situations that might arouse it. But emotion treated as a single function is as unproductive for the evaluation of personality as is memory or imagination for the evaluation of intelligence. We must devise ways and means to explore how emotions mesh into action and what role they play in personality organization.

To predict how energetic or purposeful a man is in his daily life, we must know what his values are and whether his emotions make it easy or difficult for him to adhere to these values. To predict a man's actions, we must know his purposes as well as his intelligence. Even a deceptively simple problem, like the prediction of school success, depends as much if not more on the student's motivation as on his intelligence—hence the search for "nonintellectual factors" of college success. To discover what a man's achievement is going to be and whether college success is something he is willing to work for, we must know what his attitudes are, how he thinks success can be obtained, whether he thinks it worth while, and what is worth while in life for him. Emotions will enter into such attitudes, but measuring his emotions or describing them will not give us the slightest clue as to how far they influence his actions, just as knowing the quality or intensity of a man's memory or imagination will give us no indication of his intelligence. To evaluate motivation, we have to measure intention and emotion-in-action, just as we in fact measure memory and imagination-in-action in intelligence tests. Emotion-in-action means emotion controlled and directed, just as imagination-in-action or memory-in-action means directed and controlled imagination or memory. To be sure, goal-directed action includes both emotion and imagination, in fact, it includes every single psychological function. But the performance from which intelligence is inferred includes little emotion and a great deal of imagination and memory, while sustained purposive activity depends primarily on a man's motives and his well-directed emotion.

The best kind of test for evaluating human motivation seems to be a storytelling test, whether it is the TAT, MAPS, BLACKY, or a similar test. In telling a story, the individual is forced to fit emotion into action, and to do it in a way that agrees with his attitudes and convictions. To fragment the story by counting themes or rating the intensity of various emotions is to throw away the only chance we have of gaining a working knowledge of a man's principles of action. Such a procedure would be comparable to scoring the number of times a child says the word "ball" in Binet's ball and field test and disregarding the path he maps out for finding the ball in the field.

We have indicated earlier in the Appendix how the TAT stories can be scored so as to preserve the motivational pattern that reveals the principles a man lives by. If personality, in Gasson's definition, is "the patterned totality of human powers, activities and habits, uniquely organized by the person in the active pursuit of his self-ideal" (1954, p. 219), it is revealed not only in behavior generally, but in the TAT in particular. The scoring system that can measure this pattern is still being developed. We are but at the beginning of a research program in this area which we hope will prove of interest to many others. Perhaps combined effort will finally yield a measure of personality and its motivation that is similarly useful, even though similarly restricted, as are our present tests of intelligence.

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